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HEMANGIOMA OF THE UTERUS TREATED WITH ROENTGEN RAYS*

Report of a Case

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BROOKLINE

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UNUSUAL lesions often stimulate great interest and occasion much speculation as to treatment. It is with this in mind that a case of hemangioma of the uterus is presented. This lesion is rare. It was encountered only once in over 1100 consecutive hysterectomies in the Peter Bent Brigham Hospital during a period of twenty-five years. A review of the literature is given by Horgan,[1] who in 1930 reported 1 case, together with 20 collected from the literature. Sixteen additional cases have been reported[2-13] making a total of 38 cases on record.

CASE REPORT

A 34-year-old married white woman was admitted to the Peter Bent Brigham Hospital on August 17, 1931, complaining of profuse vaginal bleeding of 3 months' duration. The previous menstrual history presented no abnormal episodes. The menarche occurred at 15 years of age, with subsequent catamenia of 3 to 4 days' duration every 28 days. There had been no menorrhagia, metrorrhagia or dysmenorrhea. The only pregnancy was terminated by a normal uncomplicated delivery of a full-term infant, 3 years before entry. Other than an attack of lobar pneumonia, complicated by empyema and necessitating a right thoracotomy 2 years before entry, the patient had always been in excellent health.

The last normal period occurred 4 months before entry. The following period, 1 month later, was prolonged to 10 days, but the daily bleeding was normal in amount. On June 1, 1931, 2 months before admission, the patient had a profuse menstrual period of 10 days' duration. From that time until her admission she bled persistently, with frequent, profuse uterine hemorrhages. Associated with this bleeding, she had been aware for 3 months of a mass in the lower abdomen, progressive weakness and a heavy dragging sensation in the pelvis.

On physical examination the patient was found to be a well-developed and well-nourished pallid woman. There was a well-healed thoracotomy scar on the chest. The lungs were resonant throughout, with no evidence of residual disease. The heart was of normal size, and the sounds were of good quality with normal rate and rhythm. The blood pressure was 110/60. The abdominal examination was negative except for the presence of an irregular, firm mass rising about 5 cm. above the symphysis in the midline. On pelvic examination the suprapubic mass was contiguous with a normal cervix and was freely movable without tenderness. The adnexa were normal.

Laboratory studies revealed grossly bloody urine (voided specimen), with a specific gravity of 1.024, a very slight trace of albumin and sediment containing innumerable red blood cells. The white-cell count was 5900, the hemoglobin 60 per cent (Sahli) and the red-cell count 3,216,000. Blood Hinton and Wassermann tests were negative.

With a preoperative diagnosis of fibroid uterus, on August 18, 1931, under Avertin-ether anesthesia, a supracervical hysterectomy, bilateral salpingectomy, left oöphorectomy and incidental appendectomy were done. The uterus was irregular, firm in most part, but showed some definite softening on the posterior wall, which was assumed to be a degenerating fibroid. The uterus was amputated at its junction with the cervix, and the cervix closed with interrupted sutures. There was not sufficient reflected peritoneum, however, to cover the raw surface of the cervical stump; the omentum was brought down to the operative field, but was not fixed to the cervix. The wound was closed in four layers.

The postoperative course was uneventful. There was no vaginal bleeding, the urine showed only a rare red blood cell by the 7th postoperative day, and on discharge on the 17th postoperative day the cervix was well suspended and the parametrial tissue normal.

Pathological studies of the removed specimen showed a moderately enlarged uterus, 12.0 by 12.5 cm., with several nodular masses bulging through the external surface which were smooth with reddish-purple, well-circumscribed discolorations. On opening the uterus the endometrial cavity was seen to be displaced well to the left, and bulging into the lumen there were several large, purplish-red polypoid masses which extended down to the external os. The remainder of the endometrium was smooth and of normal color. The cut surface of the myometrium presented small, irregularly shaped, well-circumscribed, purplish-red areas, and some brownish-red areas which were hemorrhagic in appearance. Though the cut surface bulged slightly, there were no fibroids. Both tubes were normal in appear-

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ance. The ovary was cystic and only slightly larger than normal, the cysts containing clear fluid.

The histological examination of stained tissue showed a diffusely invading hemangioma with many intravascular extensions. The section taken through the site of amputation showed that the tumor had not been completely removed. The increased thickness of the uterine wall was due largely to invasion by the hemangioma, which spread apart the fibromuscular bundles of the myometrium. There was no sclerosis of the tumor elements. Small hemorrhages were seen in the large, solid masses of minute

FIGURE 1.

Photomicrograph showing extension of the hemangioma into the myometrium and an intravascular extension below and slightly to the right. Eosin-methylene-blue stain (× 115).

endothelial-lined spaces which composed the tumor. The tubes and ovary were normal microscopically.

The postoperative convalescence was uneventful. The patient was seen in the Outdoor Department, where a cauterization of the cervix was performed which effectively controlled a low-grade endocervicitis. There had been no vaginal bleeding, and no symptoms referable to the pelvis. Nine months after the operation the patient began to suffer frontal headaches, which became progressively worse and almost constant. A pelvic examination 1 year after the operation showed a marked increase in the size of the cervix and a contiguous fixed mass about 15 cm. in diameter about it. The patient was readmitted to the hospital with the diagnosis of recurrent hemangioma and possible cerebral metastases. Examination of the eye grounds revealed a questionable choking of the left disk. In other respects the examination was negative. Right stereoscopic anteroposterior and posteroanterior roentgenograms of the skull showed no localizing signs of tumor or disease. Stereoscopic films of the chest gave no evidence of metastases.

In view of the absence of signs of metastases, removal of an obvious recurrence seemed indicated. The patient was therefore subjected to an exploratory laparotomy on August 6, 1932. On opening the abdomen a round, bluish fluctuating cystic tumor was encountered which filled the basin of the pelvis. An exploration of the upper abdomen showed a normal liver and no evidence of extension or metastases. There were multiple omental adhesions. On releasing these adhesions it was found that the tumor lay between the layers of the mesorectum and mesosigmoid, having pushed itself up out of the pelvis between these layers. It was widely attached by a broad base which apparently sprang from the left aspect of the stump of the cervix. It appeared that in endeavoring to free the tumor there was grave risk of injuring the blood supply of the bowel, since there was no line of cleavage. The tumor was firmly fixed against the left lateral pelvic wall, as well as to the base of the broad ligament and the right side of the cervix. It was evident that the tumor was invading the contiguous structures. After prolonged dissection, involving a great deal of bleeding, it was concluded that the lesion was inoperable. Toward the base of the cyst there were a number of rounded, elastic, bluish or purplish tumor masses sprouting into the lumen of the main cyst. A specimen from this area was taken for biopsy.

The microscopic examination of the tissue from the cyst showed the tumor to be composed of masses of innumerable tiny vascular channels, small and irregular sinusoids and numerous small blood vessels, lined by medium-sized to small-sized endothelial cells which had relatively large nuclei, ovoid to fusiform in shape. The angioma was slowly growing, but in occasional areas appeared to be invasive in type with frequent intravascular extensions.

Following this second operation the patient did well. In the hope that the lesion might prove radio-sensitive, roentgen-ray therapy was instituted. On the 12th postoperative day the patient received the first of a series of six high-voltage roentgen-ray treatments of approximately 400 r each, three anteriorly and three posteriorly to the pelvis. Each treatment consisted of a 20-minute exposure over an area 30 by 30 cm., with 0.5 mm. copper and 1.0 mm. of aluminum filtration and a target-skin distance

FIGURE 2.

Photomicrograph showing intravascular extension of the tumor in a section of the myometrium removed at the first operation. Eosin-methylene-blue stain (× 230).

of 40 cm. The last treatment of the series was on August 27, 1932. She was discharged on the 21st postoperative day, August 27, with the wound well healed and the cervix well suspended and fixed. At discharge, there was a fixed, firm mass 7 cm. in diameter contiguous with and above the cervix.

The patient was followed in the Outdoor Department. She was symptom-free except for a transient attack of edema of the right leg following discharge.

Following a similar series of high-voltage roentgen-ray treatments 2 months after operation, a rectal examination showed the mass still present above the cervix. The cervix was small and fixed, with smooth induration of the bases of both broad ligaments. After a third series of six roentgen-ray treatments of 400 r each, 5 months postoperatively, a pelvic examination showed the supracervical mass had disappeared, the firm smooth thickening of the bases of the broad ligaments was unchanged, and the cervix was still fixed. The body weight was 145 pounds.

A final course of six roentgen-ray treatments was given 15 months postoperatively. Other than some transient edema of the left leg following these treatments, there were no complications. The patient continued in good health with no complaints, maintaining her weight and living a perfectly normal life.

When seen in August, 1938, 6 years after the second pelvic operation, the patient appeared well and did not complain of discomfort. The body weight was 144 pounds. The lungs were resonant throughout. Abdominal examination showed a diffuse area of fibrosis and edema of the

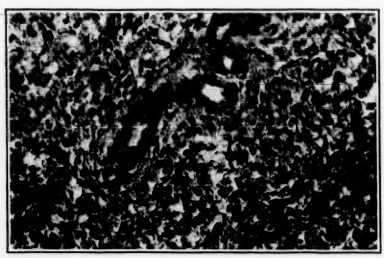

FIGURE 3.

Photomicrograph showing a portion of the recurrence of the hemangioma, which was removed at the second operation. Eosin-methylene-blue stain (× 210).

suprapubic skin, with many of the typical telangiectases which are seen following high-voltage roentgen-ray therapy. This area extended upward for a distance of about 10 cm. Similar telangiectases were noted on the buttocks, but edema and fibrosis were absent. Pelvic examination showed the small, firm cervix to be high in the vaginal vault, fixed between the markedly thickened bases of the broad ligaments. No masses could be felt. Stereoscopic roentgenograms of the thorax and pelvis showed no evidence of metastases to lungs or bone and no tumor masses.

While hemangioma of the uterus is a rare condition, the ability to recognize the lesion when encountered will aid the surgeon materially in carrying out the form of treatment that offers the best chance of cure. Of the cases reported relatively few have been associated with alarming symptoms. However, the case herein presented and the cases reported by Siegel,[16] Reder,[17] Horgan,[1] Boldt,[18] and Boks[19] were associated with serious hemorrhage, making surgical intervention imperative. In 2 cases — that reported by Pantzer[15] and the one reported above — there was severe hematuria. It is obvious that many other pathologic lesions would produce the same symptoms. The diagnosis, then, can be made only at operation. Any spongy, soft, purplish areas or tumors which exude old blood should arouse suspicion.

Hysterectomy has been a satisfactory procedure for treating those cases in which the tumor has not involved other pelvic organs. However, as demonstrated by this case, complete extirpation of the uterus must be done if the tumor involves the cervix. In the treatment of uterine hemangiomas that involve pelvic structures beyond the confines of the myometrium, it is apparent that the danger of hemorrhage and severe injury to the involved organs presents a difficult problem if surgical removal of the tumor is attempted. Ligation of the vessels, individually or en masse, does not preclude the possibility of secondary hemorrhage or recurrence. The successful outcome of this case establishes the fact that the use of high-voltage roentgen-ray therapy is the treatment of choice for these rare but extensive pelvic tumors. Hemangioma of the uterus, therefore, must be added to the list of radio-sensitive tumors in the pelvis.

SUMMARY

A case of hemangioma of the uterus is presented, with a successful result following deep roentgen-ray therapy.

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THE PREVENTION AND CONTROL OF TUBERCULOSIS IN MASSACHUSETTS*

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THE trend in the death rate from tuberculosis fortunately continues downward. In the United States for the year 1938 the rate was 48.6 per 100,000 and in Massachusetts 34.7 for the pulmonary and 3.1 for the extrapulmonary forms of tuberculosis, or 37.8 for all forms of the disease. For 1939 in Massachusetts the rate for the pulmonary form of the disease was 33.8, for extrapulmonary forms 2.2, or 36.0 for all forms.

TUBERCULOSIS IN MASSACHUSETTS

It is important to present the tuberculosis situation in Massachusetts somewhat more in detail and to consider its bearing on the prevention and control of the disease. I am indebted to Dr. Philip E. Sartwell,[1] assistant director, Division of Tuberculosis, Massachusetts Department of Public Health, for the statistical data on death rates, facilities and costs.

Tuberculosis is still a major public-health problem in Massachusetts and in 1939, with 1602 deaths, ranked seventh as a cause of death; but this number of deaths, large as it is, does not serve to measure the full significance of the disease.

Tuberculosis is the leading cause of death in the State at the most productive period of life. More persons between nineteen and thirty-five years of age succumbed from tuberculosis than from any other single cause in 1938. The death rate from the pulmonary form of the disease in this age group rises to about 40 per 100,000 population and tends to rise still higher in the remaining years of life.

The relative importance of the pulmonary form of the disease is increasing and that of the extrapulmonary form diminishing. In 1921, deaths from pulmonary tuberculosis comprised about 83 per cent and those from extrapulmonary forms about 17 per cent. In 1939, the figures were 94 and 6 per cent respectively. Though extrapulmonary tuberculosis is frequently secondary to pulmonary lesions, infected milk is a factor. Elimination of the disease in cattle and pasteurization of milk are largely responsible for the diminishing proportion of deaths from extrapulmonary tuberculosis. To eliminate the chance of human infection from tubercle bacilli or other organisms in milk, all milk should be pasteurized.

The spread of tuberculosis is more readily prevented in institutions than in the home, and it is of interest to note that in 1938 about 80 per cent of the deaths from the disease occurred in institutions and only about 20 per cent in the home. For the benefit of the individual, and the protection of the family and the community, patients with tuberculosis should be urged to go to institutions.

The degree to which tuberculosis is a menace is not the same in all counties in the State. In general, those counties having the highest density of population have the highest death rates. Over a five-year period (1935-39), excluding the counties of Dukes and Nantucket because of their small populations, Suffolk County had the highest death rate, 57.2, and Franklin County the lowest, 20.1.§

FACILITIES

The facilities in 1939 for patients with tuberculosis in Massachusetts comprised 4522 beds in federal, state, county, municipal and private institutions, a ratio of beds to deaths in 1939 of 2.8:1.

Diagnostic clinics under official auspices are available throughout the State. Diagnostic outpatient departments are maintained at the state and county sanatoriums, and through extension of their facilities there are twenty-three extramural consultation clinics, for the most part located in outpatient departments of general hospitals and with x-ray facilities available.

In addition, there are thirty-nine tuberculosis dispensaries, conducted by local boards of health. X-ray facilities, though indispensable, are lacking in these dispensaries except in a few of the larger communities, and it is desirable, as recommended by the Massachusetts Health Commission (House Bill No. 1200, p. 74), that all communities except those with approved tuberculosis hospitals be encouraged to arrange with the state and county sanatoriums for tuberculosis diagnostic service and to discontinue their locally staffed clinics.

COSTS OF TUBERCULOSIS

Facilities for tuberculosis in Massachusetts were maintained at an estimated cost in 1938 of

*The presidential address delivered at the annual meeting of the Massachusetts Tuberculosis League at the Middlesex County Sanatorium, Waltham, Massachusetts, March 28, 1940.

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§Figures corrected for residence.

$5,106,238.17, not including federal contributions, expenses of local health departments in case-finding, supervision of patients in the home, maintenance of dispensaries and other related costs.

Examination of School Children

Clinics under the auspices of state and county sanatoriums for the school year 1938-39 covered a high-school population of 53,285 children, of whom 34,897 (65 per cent) were tuberculin-tested. The proportion investigated is dependent on parental consent and, strange as it may seem, it has seldom been possible to test more than about two thirds of the children.

Clinics under other auspices covered a school population in one city of 19,351, of whom 10,202 (53 per cent) were tuberculin-tested, and in another city 1215, of whom only 244 (20 per cent) were tuberculin-tested. Failure to investigate a larger proportion of the children may be taken in part as a measure of the shortcomings of the tuberculosis associations in health education.

In a total school population of 73,851 children, 45,343 (61 per cent) were tuberculin-tested and 9730 (21 per cent) gave positive reactions. In the course of the investigation, 12,503 children were x-rayed. Forty-four cases of the adult type of tuberculosis were found, or about 1 for every 1000 tuberculin-tested children and 1 for every 221 reactors. The childhood type of tuberculosis was discovered in 1083 children, or 1 in every 42 tuberculin-tested and 1 in every 9 reactors.

School Program

The routine examination of school children is an expensive project. Estimating the cost at $1.00 to $1.50 per child tested with tuberculin, the expense of finding each case of the adult type of tuberculosis in a child amounts to between $1000 and $1500.

The adult type of tuberculosis in children is serious and the outlook is poor. It is of great moment to make the diagnosis early and resort at once to appropriate treatment. The childhood type, on the other hand, is usually mild and the outlook favorable. Its discovery is to be regarded as an indication for certain precautions and further observation by x-ray.

In communities where the case-finding program both in and outside the schools is well developed and a large proportion of the contacts of all known cases are examined by x-ray, the chance of finding a previously undiscovered case of the adult type of the disease in a school child is much diminished. The per capita cost of finding such a case is, in consequence, much increased.

It would seem, therefore, that in communities where the case-finding program among contacts is well developed and funds are limited, the routine investigation of all contacts is to be preferred to the investigation of school children when a choice of methods must be made.

Investigation of Contacts

An important present responsibility of the tuberculosis associations is the promotion of the new rules and regulations of the Massachusetts Department of Public Health.[2] Under these regulations, "as soon as a diagnosis of tuberculosis has been established, arrangements should be made for the examination, including an x-ray of the chest, of all members of the immediate family and of other persons with whom the patient has been in close contact."

Examination of family and other contacts has the advantage of a higher proportion of positive findings than is obtained in other groups in the community, and offers the opportunity of benefit to the individual through the application of appropriate treatment and to the family and the community by the breaking of contact. If the family cannot afford an x-ray examination by a private physician, facilities are fortunately available through the state, county and municipal sanatoriums.

Case-Finding in Other Groups

It is desirable that certain groups be routinely investigated. Nursemaids and other domestic servants in homes where there are children should have an x-ray examination prior to employment for the purpose of protecting those with whom they come in contact. All school teachers and other employees in contact with children should be examined for tuberculosis for the protection of children.

With respect to the investigation of school teachers, attention may be called to the appeal to the Massachusetts Tuberculosis League to formulate suggestions for school departments and the recommendation adopted at the directors' meeting of the League on March 30, 1939, as follows:

> It is recommended that school authorities require, prior to employment and at suitable intervals thereafter, the examination by x-ray and other accepted procedures, of all teachers and other employees in contact with children in the public and private schools. No teacher or employee with active pulmonary tuberculosis or any form of tuberculosis which may endanger the health of the children will be permitted to teach or continue in employment. The x-ray examination shall be made at an x-ray laboratory approved by the school committee. Examination is to be made by a physician or physicians designated or approved by the school department and be free of expense to the person examined.

Arrangements may be made by the school department for such examinations by the staffs of the state or county sanatoriums.

The school boards of thirty-four communities in Massachusetts have adopted this recommendation or have a similar regulation in effect. In this connection, it may be noted that periodic examinations for tuberculosis of pupils, teachers and employees in public schools is now required by law in New Jersey.[3] Pupils who fail to comply with this requirement and pupils with tuberculosis in a communicable form are excluded from schools.

Medical students, student nurses and interns in hospitals are more intimately exposed to tuberculosis than are those in other occupations, and should in consequence be routinely examined.

From an inquiry conducted by the League relative to the practice of examination of nurses, it was found in returns from sixty of sixty-seven training schools that an x-ray examination is done annually on all students in thirty-two, and twice during the course in six. In the remaining twenty-two training schools no x-ray examination is made at any time.

In view of the increased hazard to nurses from tuberculosis and the importance of its early discovery, the Executive Committee of the Massachusetts Tuberculosis League voted to submit the following recommendation to the Executive Committee of the Massachusetts Hospital Association:

> It is desirable in the opinion of the Executive Committee that there be a chest x-ray examination annually, or oftener if necessary, of student nurses and all other hospital personnel in contact with patients, and including food handlers. The first examination should be made about the time of beginning employment.

Responsibility of Local Boards of Health

According to the rules and regulations of the Massachusetts Department of Public Health, "it is the responsibility of the local board of health to provide hospital care for cases of tuberculosis, when needed, and to see that contacts are examined where such examination cannot be made through a private physician." It is to be expected that the local tuberculosis associations can be of assistance to local boards of health in promoting that part of this regulation which has to do with the examination of family contacts.

Community Responsibility for Relief

In carrying out these regulations. economic factors must not be permitted to stand in the way of proper treatment for the individual and the protection of the family and the community. Means must, if necessary, be provided from public-welfare or social-welfare agencies adequately to meet the needs of the family occasioned by illness from tuberculosis.

In this connection, attention may be called to the principles under which the tuberculosis associations operate. It is not their function to provide relief out of Christmas Seal funds. It is their function to encourage adequate relief by appropriate agencies, but beyond this they should not go, except in an emergency and as a temporary expedient.

Responsibilities of the Tuberculosis Associations

It has seemed desirable to the Executive Committee of the League to consider how, under changing conditions, the associations can use their funds to the best advantage. A recently formulated interpretation of the pamphlet *Authorized Forms of Tuberculosis Work*,[4] has been published.[5]

The Executive Committee plans to survey the activities of the local tuberculosis associations, give attention to their special problems and, so far as possible, assist them in planning and conducting a program for the prevention and control of tuberculosis in their communities.

Detailed comment on the desirable activities of the associations is unnecessary, but certain aspects may be emphasized. The chief concern of the voluntary associations is the formulation of a well-rounded program in accord with local needs and in co-operation with official agencies. Full-time trained workers are necessary for the accomplishment of these purposes and, where lacking, should be secured. Funds for this purpose should, if necessary, be made available through a change of policy with respect to the allocation of receipts from the Christmas Seal sale. At present in many communities large proportions of these funds go toward the support of summer health camps. In some communities considerable proportions are distributed to local tuberculosis committees to supply milk for tuberculous families and school children, dental service and tonsillectomies. These funds can be used to better advantage for health education; for the promotion of case-finding, hospitalization and the breaking of contact; for rehabilitation; and for the investigation of local tuberculosis needs.

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MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FIFTY-NINTH ANNIVERSARY

THE one hundred and fifty-ninth anniversary of the Massachusetts Medical Society was observed in Boston, Tuesday and Wednesday, May 21 and 22, 1940. The headquarters were located at the Copley-Plaza Hotel where all scientific meetings were held and exhibits were shown.

First Day, May 21

A general clinical meeting was held in the Sheraton Room in the forenoon under the chairmanship of Dr. A. Warren Stearns. The attendance was 228.

The supervising censors met in Parlor A at 10:00 a.m. and the Council was convened in the Swiss Room at 10:30. The Cotting Luncheon was served at 1:00 p.m.

During the noon hour there was a series of section round-table meetings and luncheons at which the new officers were chosen (see Appendix).

The Section of Medicine met in the President's Room of the University Club; the attendance was 89.

The Section of Obstetrics and Gynecology held its meeting in the Main Dining Room of the University Club; there was an attendance of 144.

The Section of Surgery met in the Crystal Room of the Westminster Hotel, with an attendance of 50.

The Section of Pediatrics held its meeting in the Ballroom of the Westminster Hotel; the attendance was 70.

The Section of Dermatology and Syphilology met in the State Salon of the Copley-Plaza, with an attendance of 50.

In the afternoon a general clinical meeting and symposium on respiratory-tract infections was held in the Sheraton Room under the chairmanship of Dr. William H. Robey. The attendance was 388.

The annual dinner was held in the Main Dining Room at 7:00 p.m. The attendance was 264. During the dinner the President presented the newly elected officers and the guests at the head table. He next presented the delegates from the other New England state medical societies as follows:

Maine: Dr. Thomas A. Foster, Portland
New Hampshire: Dr. George V. Fiske, Manchester
Dr. Addison Roe, Newport
Vermont: Dr. Roland E. McSweeney, Brattleboro
Rhode Island: Dr. George L. Young, East Greenwich
Connecticut: Dr. Walter C. Haviland, Mansfield
Dr. Edward J. Ottenheimer, Willimantic

Following the dinner Dr. Ernest W. Goodpasture presented the Shattuck Lecture in the Sheraton Room. The subject was "Immunity to Virus Diseases: Some theoretical and practical considerations" (published in the *Journal*, issue of May 30). The attendance was approximately 575.

Second Day, May 22

In the forenoon a general clinical meeting and symposium on syphilis was held in the Sheraton Room under the chairmanship of Dr. C. Guy Lane. The attendance was 400.

ANNUAL MEETING

The annual meeting of the Society was called to order by the president Dr. Walter G. Phippen, of Salem, at 11:00 a.m. There were approximately 400 in attendance. The minutes of the one hundred and fifty-eighth meeting of the Society were approved as published in the *New England Journal of Medicine*, issue of July 6, 1939. The Secretary then presented the changes in membership as follows:

Membership reported June 8, 1939			5438
Less member who was not restored since he failed to meet his financial obligation			1
			5437
Losses			
Deaths	99		
Resignations	23		
Deprivations	38		
		160	
Gains			
New fellows	252		
Readmitted by censors	3		
Restored by Council	10		
		265	
Net gain			105
Membership reported May 22, 1940			5542

The Secretary announced that the New England Postgraduate Assembly would be held in Sanders Theatre and Memorial Hall, Harvard University, on Wednesday and Thursday, November 13 and 14, 1940. There is an impressive list of speakers. The committee in charge hopes to

make this occasion even more of a success than it has been in the past.

The President then proceeded to give his report on the state of the Society as follows:

It is my duty at this annual meeting to report on the state of the Society.

I am glad to report that it is, in a way, in a very healthy condition and for the moment sailing on a fairly even keel. The membership has increased by 105 so that it now numbers 5542. There are still a large number of physicians in Massachusetts who are not members of the Society but who should be, and I urge you who know any such to seek to enroll them.

I wish to express my very high regard for our efficient secretary, Dr. Alexander S. Begg. Without his intimate knowledge of the Society a new president would be entirely lost.

Our conscientious and careful treasurer, Dr. Charles S. Butler, reports that the financial situation of the Society is as sound as he can make it, upon a foundation of high-grade bonds. The income from our invested funds has been only slightly lower than that for the corresponding five months of 1939. All bonds except three are paying interest, and these three have been marked down by vote of the Council to nearly present values. He reports that the collection of dues by district treasurers shows some improvement over previous years. We should consider ourselves very fortunate to have such an able, far-sighted and sagacious treasurer, and it is a pleasure to thank him sincerely in your behalf.

There have been very few changes in the committee chairmen during the year. Dr. Roger I. Lee asked to be relieved of his duties as chairman of the Committee on Publications, a position he had filled with honor since 1933. This resignation was accepted with regret, and Dr. Frank H. Lahey appointed in his place.

After two hard and trying years as chairman of the Committee on State and National Legislation, Dr. Charles C. Lund felt obliged to retire. During these years he had given unstintingly of his time and energy in the interest of the Society. His resignation was accepted with great regret, and Dr. Edward L. Merritt, of Fall River, was appointed in his place. The committee has functioned energetically and well, and I wish to thank the members publicly for their unselfish effort.

The new Committee on Financial Planning and Budget under Dr. John Homans's careful leadership has, I am sure, justified its formation.

The Committee on Postgraduate Instruction successfully conducted a second postgraduate assembly in spite of strong opposition by Jupiter Pluvius. Its district courses have for the most part been well attended, and I believe serve a useful purpose.

Perhaps the thing that amazed me most on assuming office as your president was the great number of charges brought before the Committee on Ethics and Discipline. Many of them are trivial, but many have enough foundation in fact to make one pause and wonder why doctors cannot be honest with themselves. In these times of unrest it behooves us all to be overcareful in our deportment and consideration of patients and their families. Errors of commission in overcharging or unethical conduct only make the critics of the medical profession less tolerant and more expressive in voice and print.

The Council has adopted the recommendations of the Committee on Public Relations setting forth five general principles governing the establishment of projects that aim to prepay the costs of medical care. With these general principles established the special committee will conduct its studies in the hope that it can present a safe, workable and efficient set-up for the consideration of the Council at a later date. It will endeavor to secure the passage of an enabling act at the earliest possible moment.

I wish to thank the members of the Society for their patience and for their sympathetic understanding of the efforts of your officers in trying to reach a solution of this very difficult problem, and I ask your indulgence and continued patience in the months that are to come so that we may avoid the pitfalls other state medical societies have fallen into and sustain the fair name of the Massachusetts Medical Society.

I think we have only to consider the example of the Blue Cross to realize that patience, forbearance and frank discussion without rancor will straighten out many difficulties. All the controversy regarding anesthetists, pathologists and other questionable medical services has been cleared up, the organization is becoming stable and financially sound, and physicians for the most part believe it is good and has come to stay. I, for one, believe we should give it whole-hearted support.

I regret that I have been unable to visit all the district societies. This is a very pleasant presidential duty, but at times it is difficult to fulfill. Some districts plan their meetings in the fall and publish their programs. Others are not so forehanded and arrange their meetings from month to month. I strongly recommend to the officers and committees of the districts that they consider the setting up of programs for the entire season. I believe the added attendance will justify the effort. To those district societies I have been unable to visit I offer my humble apologies. My only consolation is that my loss, I am sure, has been greater than theirs.

I have been impressed with the large amount of clinical material in small hospitals that could be used for teaching purposes. It is not necessary to have a large ward service for clinical teaching. It is not necessary to have a teaching staff to use this material. Any physician can train himself to summarize an interesting and instructive case and present it to his confreres. This to my mind is a lost opportunity for postgraduate instruction. The time has gone by when fear of criticism or ridicule should deter a physician from reporting and offering for discussion his own private cases.

Several district societies incorporate a one-hour or two-hour clinical exercise in their regular meetings. This in no way interferes with a dinner or luncheon at which an authoritative speaker may be heard. Where this has been tried the attendance has very markedly increased, and the interest has been sustained. It is a benefit to those who give the clinic as well as to those who attend.

I consider this a most important contribution to postgraduate instruction and commend it to the consideration of the district society officers and committees. I should like to go farther than this and say that every hospital, large or small, should inaugurate monthly, fortnightly or even weekly clinical exercises. We have, for some years, conducted weekly clinical exercises at the Salem Hospital every Friday morning at nine o'clock, rain or shine, and they are very largely attended. These exercises may occasionally take the form of clinicopathological conferences, tumor clinics or clinics conducted by special departments, such as those of urology or orthopedics. I assure you that they are good for the hospital, good for the staff, good for the community doctor and good for the soul.

I desire to call your attention to membership in the House of Delegates of the American Medical Association.

The House of Delegates is the sole legislative body of the American Medical Association. It alone has the authority to formulate the policies of organized medicine. Your delegates should represent the well-considered views of the Society and should be elected with that thought in mind, as I believe they are and have been in the past. But looking to the future I am wondering if some consideration should not be given to their length of service.

The by-laws of the American Medical Association require that delegates be elected for a term of two years, but our by-laws do not specify how many times a delegate may be re-elected or whether he should be re-elected at all. A delegate may be elected for one term and then dropped, or he may be re-elected continuously for many terms, sometimes for twenty years or more. I am quite certain that to elect a man for one term and no more is of little value to the delegate, no value to the American Medical Association and an economic loss to the Society. I am not quite so clear that the same holds true in inverse ratio—that the longer a man serves the more valuable he becomes.

Perhaps one criticism I have of the House of Delegates is that too many delegates have been there too many years. With the growing changes in the economic and social structure of our country and the necessity to adapt our medical actions to conform with them, it seems to me that fresh points of view should be brought periodically to the House. I commend for your consideration a change in our by-laws so that, by some staggering method perhaps, delegates should automatically be re-elected for say three or four terms (six or eight years) and then be ineligible for at least one term. This would allow the installation of new blood regularly, and at the same time return a fair number of older members.

The office of president of the Massachusetts Medical Society is an increasingly exacting one. While the physical effort required is not great, the mental effort necessary to solve the many intricate problems is considerable. Particularly is this so in the early months of his incumbence. I am wondering if the time has not come to install a president-elect, who would use his pre-presidential year learning at close range something of his duties for the year to come. If this were done the tenure of office might well be reduced to one year. The president-elect could take over the pastoral visits to the district societies and be in a better position to understand their problems when he becomes president. He could also take over other assignments. In this way the president would arrive at his position well versed in Society matters.

The vice-president's office cannot be put to good use because of our tradition of re-electing the president for a second term and not re-electing the vice-president. I believe this should receive your serious consideration.

The National Health Program continues to move slowly on at Washington. The Wagner Bill so bizarre in outline and overwhelmingly costly in operation remains in committee. Many other bills have been introduced into the Senate and are grouped together with the Committee on Education and Labor. Only one has emerged. The so-called Wagner-George Hospital Bill has been reported out and apparently unanimously and with a strong recommendation for its passage. This bill provides for the construction of hospitals where need for them is shown, this need to be determined at least during the first year of the operation of the bill by a National Advisory Council to consist of the Surgeon General and eight members appointed by the Surgeon General with the approval of the Federal Security Administrator. "The eight appointed members shall be selected from leading medical and scientific authorities who are outstanding in matters pertaining to hospitals or other public services." This bill incorporated some of the suggestions made by the American Medical Association and the American Hospital Association and for the first time, as noted above, recognizes medical experts in advisory capacity. It is encouraging in these respects. It puts a heavy responsibility on the Surgeon General in choosing his advisory council. In the original Wagner-George Hospital Bill the title to the hospital was to remain with the government. In this bill the hospital, after its construction, is leased to the community, but the title is to be conveyed eventually to the applicant. This apparently does away with threat of a string of government-owned hospitals. The action by the Senate will be awaited with interest.

In closing let me thank you all for the honor you have conferred upon me and for your hearty co-operation and support of my modest efforts in behalf of the welfare of the Massachusetts Medical Society.

The business meeting was adjourned and the President presented the orator, Dr. William Jason Mixter, of Boston, who spoke on "New England, Neurosurgery and the Neurosurgeon" (published in the *Journal,* issue of May 23). Following the completion of the Annual Discourse, the annual luncheon was served in the Swiss Room.

On the afternoon of the second day a general clinical meeting and symposium on sulfanilamide was held in the Sheraton Room under the chairmanship of Dr. Reginald Fitz. There were at least 600 in attendance.

The program finished at approximately 5:00 p.m.

ALEXANDER S. BEGG, *Secretary.*

* * *

The commercial exhibits numbered seventy-two and were well patronized. The scientific exhibits numbered eighteen and were of a high order of excellence. The motion-picture programs extended over the two days of the meeting and elicited much favorable comment. It is hoped that more room will be provided for this feature of the program in the future.

The Ladies' Committee was made up as follows: Mrs. Walter G. Phippen, chairman; Mrs. J. Frank Donaldson, Mrs. Thomas H. Lanman, Mrs. Roger I. Lee, Mrs. Charles C. Lund, Mrs. Frank W. Marlow, Jr., Mrs. Donald Munro, Mrs. Edward L. Peirson and Mrs. William B. Robbins. An attractive program was arranged, which on Tuesday, May 21, consisted of visits in the afternoon to the Gore House and Mrs. Arthur Lyman's garden in Waltham, dinner at Horticultural Hall and attendance in the evening at a "Pop" Concert in Symphony Hall. On Wednesday, May 22, there was a trip to Salem, which included visits to the Peabody Museum, the Essex Institute, the Pingree

House, a Salem garden and luncheon at the Salem Country Club.

The Golf Tournament, under the chairmanship of Dr. William T. O'Halloran, was held at the Belmont Country Club on Tuesday, May 21. There were twenty-two players. The distribution of prizes was as follows:

Low net (Burrage Bowl) — Dr. Oliver A. Lothrop
Second low net — Dr. John W. Henderson
Third low net — Dr. Thomas J. Cavanaugh
Low gross — Dr. Richard S. Nugent
Second low gross — Dr. Robert Dutton
Third low gross — Dr. Joel M. Melick

The total registration for the two-day meeting was as follows:

Physicians	1461
Ladies	200
Total	1661

The lists of admissions and deaths are appended, together with the official lists of officers, standing and special committees, councilors, censors and so forth.

APPENDIX

Admissions Recorded From June 8, 1939 to May 22, 1940

YEAR OF ADMISSION	RESIDENCE	MEDICAL COLLEGE
1939	Adams, Ralph Hudson, Cambridge	11
1939	Aieta, Joseph, Jr., Dorchester	10
1940	*Allendorf, Francis Jasper, Walpole	6
1940	Alpert, George Roland, Brookline	45
1940	Altschule, Mark David, Boston	11
1939	Anderson, Leonard Edward, Longmeadow	38
1938	†§Arey, Jean McIntosh, Gardner	12
1940	Arnold, Jesse Oglevee, 2nd, Worcester	2
1939	Aufranc, Otto Elmo, Brookline	11
1940	Baldwin, Albert Kilburn, Taunton	46
1940	Baldwin, Arthur Dwight, Wellesley	11
1940	Baldwin, Gertrude W., Cambridge	47
1940	Barbour, Charles Manson, Jr., Newton Highlands	9
1939	Barker, Robert Howard, Waban	11
1940	*Beer, Peter Johann Otto, Waltham	48
1939	Beigelman, Herman, Roxbury	12
1940	*Benda, Clemens Ernst, Wrentham	49
1940	Benson, Harry Ludvig, Winchester	10
1940	Berezin, Martin Arthur, Harding	10
1939	Bergman, Macks Leonard, State Farm	7
1939	Bersack, Solomon Rodney, Springfield	3
1939	Blaisdell, John William, Lawrence	12
1940	Blaney, Cyril Arthur, Westford	10
1939	*Bloom, Philip, Somerville	8
1940	Blouin, Alfred Rodolph, Northbridge	10
1940	Blum, Max, New Bedford	50
1940	Bonner, Hugh Joseph, Jamaica Plain	9
1939	Bradley, Joseph Augustine, Lawrence	12
1939	Brenner, Charles, Brookline	11
1939	Brinegar, Willard Clouse, East Gardner	16
1939	Brines, John Kincaid, Wellesley	11
1939	Bryant, Mason David, Jr., Lowell	12
1939	Buddington, Weston Turner, Newton	11
1939	Bush, Charles William Jr., Milton	10
1939	Caia, Pasquale Conrad, Springfield	12
1940	Cannon, Bradford, Cambridge	11
1940	*Canzanello, Vincent Joseph, Everett	6
1939	Carbone, Joseph Alfred, Springfield	15
1939	Carleton, Thomas McMaster, Brookfield	12
1940	*Carmody, Robert Fulton, Brookline	6
1939	Cass, Leo Joseph, Cambridge	11
1940	Chafetz, Max, Dorchester	12
1939	Cheever, Francis Sargent, Wellesley	11
1939	Chiminello, Frank Joseph, Quincy	10
1940	Chippendale, Francis David John, Fall River	19
1940	Clarke, Samuel Tracy, Boston	11
1939	Cogan, Michael Aaron, Holyoke	14
1939	Coggeshall, Howard Cranor, Boston	36
1939	Cohen, Jack Dexter, Brookline	12
1939	Cohen, Samuel Louis, Boston	10
1940	*Colantino, George Joseph, Malden	6
1939	Colcock, Bentley Prescott, Jamaica Plain	22
1939	Collins, James Francis, Gloucester	12
1939	Colman, Joseph Harry, Mansfield	12
1940	Comanduras, Peter, Roxbury	12
1940	Commette, Joseph Paul, Beverly	10
1939	*Condo, Annunziato, Lawrence	31
1940	*Conroy, John Albert, Newton	40
1939	Constantian, Harold Martin, Worcester	24
1939	Conway, Christopher Clarence, Malden	12
1939	Cooper, Philip, Brighton	11
1939	Corcoran, John Joseph, Lowell	12
1940	Crandon, John Howland, Boston	11
1939	Crowley, John Joseph, Lynn	11
1940	Cutler, Joseph George, Salem	18
1939	Damiani, Clito Robert, Lowell	12
1940	Davis, Burnet Madura, Boston	11
1939	†Dawson, Raymond J., Methuen	9
1939	Dee, John Foley, Indian Orchard	10
1939	DeNuccio, Adolph Alfred, Lawrence	10
1939	DerHagopian, Ardashes Paul, Chelsea	12
1939	Dickson, Ellsworth Joseph Murray, Needham	12
1939	Dineen, John Bernard, Springfield	19
1940	†Donaghy, George Everett, Boston	12
1940	*Driscoll, Daniel Timothy, Malden	6
1939	Duggan, George Leonard, Lowell	19
1939	Durant, Richard Church, Great Barrington	11
1939	Dynes, John Burton, Boston	11
1940	Elder, Burton F., Hingham	51
1939	Elia, Andrew Dimitri, Boston	10
1940	Ficicchy, John, Jr., Hyde Park	10
1939	Fiman, Charles Elden, Taunton	28
1940	Fine, Jacob Harold, Beverly	12
1940	Finley, Knox H., Brighton	1
1940	*Fischer, Joseph, Brighton	52
1940	*Fishgal, Maurice Charles, South Boston	6
1939	Flo, Spencer Carlyle, Greenfield	38
1939	Forbes, Anne Pappenheimer, Milton	29
1940	Freeman, Rowland Godfrey, Jr., Dover	29
1939	Friedberg, Isadore Hirsh, Brighton	12
1940	Friedman, Emerick, Waltham	53
1940	Fritz, Lewis Edward, Stoughton	12
1939	Fromer, John Lewis, Brookline	4
1939	Frothingham, Joseph Rotch, Boston	11
1940	*Fulder, Hans, Worcester	54
1940	Gahm, Irvin George, Boston	12
1940	Gall, Edward Alfred, Cambridge	50
1939	Gariépy, Alonzo Joseph Asias, Barre	12
1940	Gates, Donald Coats, Boston	11
1939	Gibson, John Graham, II, Cambridge	11

1939 Giddon, Elliot David, Brookline 10
1940 Goldman, Max, Boston Harbor 12
1940 Gordon, Sydney Raphael, Worcester 12
1939 Gorney, Arthur Joseph, Chestnut Hill 12
1940 Gould, David Marshal, Boston 11
1939 Grace, Sydney, Cambridge 10
1939 Grainger, James Eldredge, Worcester 12
1940 *Grinold, John Joseph, Belmont 6
1940 Gudas, Peter Paul, Boston 10
1939 Haddad, Arthur Kalil, Worcester 12
1940 Hadler, Arthur Joseph, Boston Harbor 11
1939 Haimes, Solomon Manuel, East Gardner 21
1940 Hand, Leo Vincent, Wellesley Hills 2
1940 Hawkins, Ralph Leslie, Boston 11
1940 Hearne, Thomas Myers, Melrose 10
1940 Hindman, Daniel Harold, Roxbury 11
1940 Hinds, Charles Benjamin, Jr., Worcester 11
1939 Hirtle, Ralph Benjamin, Malden 11
1939 Hiscock, Mabelle Caroline, Roxbury 18
1938 †‡Hitchcock, James, Fitchburg 63
1939 Holleran, Harold, Westfield 12
1939 Hooper, Langdon, Wellesley Hills 11
1939 Houle, Emile Antonio, Lowell 10
1940 Hoxie, Thomas Barr, Boston 12
1939 Huber, William McPherson, Brookline 22
1940 Hyder, George Abdallah, Lawrence 12
1939 Hyman, Mayer, Boston 15
1939 *Jacobs, Perry Harry, Hudson 6
1939 Janeway, Charles Alderson, Weston 18
1939 Johnson, Paul Emanuel, Attleboro 12
1939 Joy, Genevieve Louise, Stoneham 28
1940 Kaufman, Manuel, Tewksbury 12
1940 Kaufman, S. Harvard, Worcester 13
1940 Keane, Geoffrey Patrick, Fall River 11
1940 Kemble, Robert Penn, Worcester 21
1940 *Kevorkian, John Jacob, Watertown 20
1940 King, Marion Alden, Northampton 10
1940 Kinsey, Dera, Brookline 41
1939 Klainer, Max Joseph, Stoneham 10
1940 *Kotarski, William C., Springfield 6
1939 Kunkel, Paul, Boston 17
1939 Lamb, Gordon Richard, Brookline 38
1940 Lappin, Abraham Henry, Dorchester 12
1939 Lathrop, Frank Damron, Brookline 38
1939 Laughran, James Frederick, Lowell 12
1939 *Laurin, Theophile, Lowell 23, 26
1940 Leadbetter, Wyland Fenway, Brookline 18
1939 *Lentino, Joseph Webster, Clinton 27
1940 *Lever, Walter Frederick, Boston 55
1940 *Levison, Carl, Dorchester 6
1940 Lewis, Emil Harold, East Boston 10
1939 Lewis, James, Westfield 11
1939 Lindberg, Theodore Frederik, Dorchester Center 25
1939 *Lindemann, Erich, Boston 30
1940 Lowd, Harry Mosher, Jr., Salem 5
1939 Lulow, William Victor, Scituate 12
1940 MacKillop, John Angus, Cambridge 12
1939 MacLaughlin, Charles Harland, Everett 12
1940 *Maiuzzo, Anthony M., Beverly 6
1940 Malamud, William, Worcester 9
1940 *Maloof, Frederic, Boston 6
1939 Manganelli, Charles Vincent, Lynn 12
1939 Manley, James Sylvester, New Bedford 12
1939 Markey, Hilda, Allston 32
1940 †Marvin, Frank William, West Newton 11
1939 *Mattia, Anthony Francis, Fitchburg 20
1939 Mazer, Mendel, Springfield 29
1939 McCarthy, Ralph Philip, Peabody 19
1940 McLaughlin, Joseph Aloysius, New Bedford 12
1939 Mehan, Mona Catharine, Hathorne 44
1940 Meltzer, Adolph, Worcester 33
1940 *Mindus, Walter Valk, New Bedford 62
1940 Moore, Paul Thomas, Milton 11
1939 *Moriarty, James Edward, Roslindale 6
1940 Moritz, Alan Richards, Waban 16
1939 Morse, Frank Peele, Jr., Jamaica Plain 12
1939 Mulhern, John Francis, Jr., Taunton 12
1940 Mullaney, Owen Christopher, Dorchester 10
1940 Mushlin, Harry Ralph, Brookline 10
1940 *Nadel, Frederick Philip, Cambridge 6
1940 Nathanson, Ira Theodore, Boston 25
1940 Nelson, Roy Walter, Attleboro 10
1940 Newton, Aaron Wayne, Boston 12
1939 Nicholson, Morris John, Roxbury 39
1939 O'Brien, Thomas Emmet, Lynn 12
1939 O'Brien, Thomas Joseph, Hudson 12
1939 *Oddy, Parkinson Louis, Methuen 6
1939 Olans, Sidney, Somerville 10
1939 *O'Neill, John James, Salem 40
1939 Orlov, Samuel, Wareham 12
1940 Osgood, Rudolf, Belmont 15
1939 Partington, Philip Raymond, Great Barrington 29
1939 Pekala, Joseph Gabriel, Northampton 7
1940 Pelton, Thurlow Hemsworth, Pittsfield 11
1940 *Pett, Morris Hirch, Gloucester 6
1939 Philbrook, Frank Randolf, Randolph 10
1940 Pittman, Merry Elizabeth, Roxbury 15
1940 *Pollák, Otakar Jaroslav, Allston 56
1940 *Rak, Ian Parley, Lexington 57
1939 Remy, Sylvio Bernard, Webster 19
1939 Reuter, Robert John, Roxbury 35
1939 Risman, Joseph, Lynn 7
1939 Romano, John, Boston 35
1939 Rosenheim, Frederick, Belmont 29
1940 Rozanski, Frank Stanley, Worcester 58
1939 Salter, William Thomas, Milton 11
1940 Saphir, Nelson Robert, South Boston 17
1939 Sargent, Morgan, Quincy 1
1939 Schultz, Philip Ephraim, Roxbury 37
1939 *Scola, Joseph Anthony, Worcester 6
1940 Sellman, Priscilla, Newton 10
1939 Senecal, Alphonse Leonard, Beverly 9
1939 Sewall, Kenneth Wilkins, Boston 11
1939 Shlossberg, Frank, Lowell 42
1940 Shriber, William Jacob, Dorchester 38
1939 *Silbert, Nathan Ernest, Dorchester 40
1940 *Skrabucha, William Albert, Lowell 6
1939 Slemons, Marion Lucile, Quincy 38
1939 Smith, Edwin Andrew, Lynn 12
1939 Souders, Carlton Remsberg, Jamaica Plain 11
1940 Spira, Bertram, Worcester 59
1939 Staples, Oscar Sherwin, Hyde Park 11
1940 Stearns, Carl, Boston Harbor 12
1939 Steele, George Chapin, West Springfield 11
1940 Stevenson, Charles Summers 18
1939 Sullivan, Francis Xavier, Hathorne 12
1939 Sullivan, Garrett Leo, Jr., Cambridge 11
1940 Sweetsir, Frederick Nelson, Roxbury 12
1940 Tate, John Comstock, Westfield 60
1939 Teahan, William Waters, Holyoke 22
1940 *Thomas, John Donoclift, Nahant 20, 54
1940 Thompson, Richard Hildreth, Boston 11
1939 Thornton, Joseph Peter, Dorchester 10
1940 Toumey, James William, Jr., Dedham 29
1940 Tuck, Herbert A., Fitchburg 12
1940 Twinam, Claire Willard, Pittsfield 61
1939 Vance, Lewis Alexander, Cambridge 43

1939	Vastine, Mary Frances, Roxbury	44
1939	*Vohr, Dorothy Larrison, Lee	6
1940	Warbasse, James Peter, Jr., New Bedford	24
1939	Warren, Richard, Boston	11
1940	Warthin, Thomas Angell, Boston	11
1939	Wasserman, Mitchell, Mansfield	10
1940	Watkins, Arthur Lancaster, Arlington	11
1939	Weintraub, David, Brighton	9
1939	*Wekstein, Abraham Jacob, Mattapan	6
1939	Whelan, Edmund Lawrence, Malden	12
1939	Wiener, Harry Julius, Revere	38
1940	*Willgoose, Dorathea May, Needham	6
1939	Wilson, Carl, Springfield	43
1940	*Wissing, Egon Georg, Boston	49
1939	Wolf, William, Lowell	12
1940	Wright, Marian Lucia, Boston	10
1939	Wright, Rebekah, Arlington	25
1939	*Zalvan, Jacob, Millis	6
1940	Ziegler, Edwin E., Chelsea	34
1940	Zoll, Paul Maurice, Boston	11

Total number new fellows admitted November, 1939	138
Total number new fellows admitted May, 1940	114
Total number fellows readmitted November, 1939	1
Total number fellows readmitted May, 1940	2
Grand total	255

*The candidate's diploma was approved by the Committee on Medical Education and Medical Diplomas, and he or she was given a personal interview by this committee and permitted to take an examination before a board of censors.

†Readmitted by censors.

§Omitted from previously published list.

KEY TO MEDICAL COLLEGES

1. Yale University School of Medicine
2. Temple University School of Medicine
3. University and Bellevue Hospital Medical College
4. New York University College of Medicine
5. University of Rochester School of Medicine
6. Middlesex University School of Medicine
7. University of Vermont College of Medicine
8. Missouri College of Medicine and Science
9. McGill University Faculty of Medicine
10. Boston University School of Medicine
11. Harvard Medical School
12. Tufts College Medical School
13. University of Wisconsin Medical School
14. Vanderbilt University School of Medicine
15. Rush Medical College of the University of Chicago
16. University of Nebraska College of Medicine
17. Washington University School of Medicine
18. Johns Hopkins University School of Medicine
19. Georgetown University School of Medicine
20. College of Physicians and Surgeons, Boston
21. Jefferson Medical College of Philadelphia
22. University of Pennsylvania School of Medicine
23. Baltimore University School of Medicine
24. Long Island College of Medicine
25. Northwestern University Medical School
26. University of Bishop College Faculty of Medicine
27. Royal University of Rome
28. College of Medical Evangelists
29. Columbia University, College of Physicians and Surgeons
30. University of Giessen, Germany
31. Royal University of Medicine and Surgery, Naples
32. University of Toronto Faculty of Medicine
33. Cornell University Medical College
34. George Washington University Medical School
35. Marquette University School of Medicine
36. Indiana University School of Medicine
37. Creighton University School of Medicine
38. University of Michigan Medical School
39. University of Maryland School of Medicine and the College of Physicians and Surgeons
40. Kansas City University of Physicians and Surgeons
41. Baylor University College of Medicine
42. Dalhousie University Faculty of Medicine
43. University of Virginia Department of Medicine
44. Woman's Medical College of Pennsylvania
45. University of California Medical School
46. Medical School of Maine
47. University of Pittsburgh School of Medicine
48. Ludwig-Maximilians-Universität, Munich
49. University of Berlin
50. Tulane University of Louisiana School of Medicine
51. Ohio State University College of Medicine
52. University of Vienna
53. University of Buffalo School of Medicine
54. University of Lausanne
55. Universities of Heidelberg, Vienna, Zurich, Hamburg, Leipzig
56. Medical School of Masaryk University of Brno
57. University of Leipzig
58. Hahnemann Medical College and Hospital of Philadelphia
59. University of Illinois College of Medicine
60. University of Tennessee College of Medicine
61. State University of Iowa College of Medicine
62. Friedrich-Wilhelms University, Berlin
63. Dartmouth Medical School

Deaths Reported from June 8, 1939 to May 22, 1940

ADMITTED	NAME	PLACE OF DEATH	DATE OF DEATH	AGE
1895	Abbe, Frederick Randolph	Boston	March 2, 1939	70
1904	Adams, Zabdiel Boylston	Brookline	March 16, 1940	65
1914	Allison, Carl Edwin	Chelsea	June 9, 1939	52
1922	†Bacon, Gorham	Yarmouth Port	March 5, 1940	84
1893	†Bailey, George Guy	Ipswich	March 30, 1940	75
1892	Baker, Frederick Herbert	Worcester	October 1, 1939	72
1898	Berg, Tekla Amalia Josefina	Lynn	June 8, 1939	70
1914	Berry, Charles Francis	Boston	October 10, 1939	68
1904	†Blenkhorn, James	Stoneham	October 8, 1939	75
1902	Borden, Charles R. C.	Brookline	November 28, 1939	65
1893	Brewster, George Washington Wales	Boston	September 26, 1939	73

1907	†Brigham, Fred Clayton	Springfield	January 22, 1940	69
1892	†Burke, James Joseph	Easthampton	November 16, 1939	79
1901	Burnham, Joseph Forrest	Boston	January 26, 1940	70
1897	Butler, George Edward	Fall River	March 8, 1940	71
1918	Callanan, Francis Jervois	Boston	April 21, 1940	45
1911	Carlisle, Frank Henry	Wellesley	July 20, 1939	60
1905	Carroll, John Joseph	Holyoke	November 19, 1939	61
1933	Cassidy, James Michael	West Springfield	October 4, 1939	35
1920	Cheney, Robert Cartwright	Boston	February 8, 1940	45
1905	Chesley, Alfred Ervan	Lawrence	October 27, 1939	63
1885	Coolidge, Algernon	Boston	August 16, 1939	79
1899	Crandon, LeRoi Goddard	Boston	December 27, 1939	66
1892	†Crocker, Benton Pulsifer	Foxboro	May 26, 1939	72
1913	Cushing, Harvey	New Haven, Conn.	October 7, 1939	70
1887	Cushman, Andrew Bernard	South Dartmouth	February 13, 1940	83
1927	Damsky, Charles	Lynn	December 3, 1939	45
1906	Davis, Ernest Leland	Springfield	September 10, 1939	67
1914 1937	Dervin, Lawrence James	Somerville	March 30, 1940	64
1937	Dexter, Smith Owen, Jr.	New York City	November 25, 1939	32
1922	Doherty, Henry Leo	Stoughton	October 9, 1939	45
1918	Emard, George Adelbert	Mansfield	August 3, 1939	57
1906	†Felch, Lewis Perley	Boston	April 2, 1940	68
1921	Fitchet, Seth Marshall	Newton Centre	September 26, 1939	52
1906	Gallagher, John Henry Connaughton	St. Petersburg, Fla.	March 19, 1940	57
1914	Gaylord, James Frederick	Springfield	June 8, 1939	49
1921	Gibson, David Howard	Cambridge	June 8, 1939	54
1897	Hallett, Edward Bangs	Gloucester	November 8, 1939	75
1890	Harding, George Franklin	Brookline	May 16, 1940	77
1903	Harrington, Michael William	Indian Orchard	November 13, 1939	66
1898	Hayes, Albert Edwin	Edgewood, R. I.	Unknown	73
1902	Hewitt, William Oakes	Boston	April 27, 1940	63
1903	Hillard, James Pearse	Springfield	January 29, 1940	62
1915	Hiltpold, Werner	Easthampton	April 5, 1940	50
1917	†Howard, Charles Tilden	Hingham	September 6, 1939	68
1920	Hughes, Frank	North Weymouth	September 13, 1939	67
1897	†Irwin, Vincent Joseph	Tampa, Fla.	June 19, 1939	84
1919	Jordan, Michael Matthew	Worcester	September 30, 1939	55
1903	Kelley, Joseph Henry Hart	Dorchester	February 18, 1940	62
1880	Kittredge, Joseph	North Andover	January 31, 1940	81
1894	†Lamoureux, Joseph Elzear	Lowell	March 16, 1940	71
1900	Lane, Walter Appleton	Milton	January 21, 1940	67
1897	Leary, William Charles	Springfield	October 13, 1939	70
1900	Legg, Arthur Thornton	Providence, R. I.	July 8, 1939	65
1935	Lobo, Joseph Paulo	Boston	November 28, 1939	62
1896	Lord, Sidney Archer	Brighton	March 30, 1940	71
1920	Loring, Benjamin Tappan	Watertown	July 29, 1939	70
1900	†Lovell, Martha Eleanor	Boston	April 22, 1940	Unknown
1913	Luce, LeRoy Alson	Boston	September 27, 1939	60
1899	Mains, Charles Frederick	Dorchester	August 17, 1939	67
1915	Mains, Herbert Llewellyn	Danvers	April 6, 1940	62
1903	Marshall, Augustus Thompson	Randolph, Vt.	Unknown	64
1915	Mathewson, Frank Weeden	New Bedford	July 6, 1939	48
1908	McIntire, George Francis	West Barrington, R. I.	January 9, 1940	69
1892	Mead, George Nathaniel Plummer	Winchester	December 14, 1939	80
1904	Moline, Charles	Sunderland	November 15, 1939	63
1890	Morse, John Lovett	Newton	April 3, 1940	75
1884	†Munro, Walter Lee	Providence, R. I.	October 23, 1939	82
1895	Noble, Anngenette Fowler	Westfield	April 30, 1940	79
1903	Nye, Harry Royal	Leominster	January 8, 1940	68
1906 1916	Oak, Charles Arthur	Lynn	November 15, 1939	60
1902	O'Brien, Walter John Leo	Jamaica Plain	May 1, 1940	66
1930	Page, Peirson Sterling	Andover	May 23, 1939	67
1891	†Payne, James Henry	Allston	January 31, 1940	77
1916 1921	Peck, Martin William	Boston	May 7, 1940	60
1899	Peirce, George Alphonso	Lowell	February 21, 1940	65
1904 1928	Pope, Ernest Folger	Boston	January 26, 1940	65

1908	Pratt, David Damon	New Bedford	January 29, 1940	59
1910	Preble, William Emerson	Boston	January 22, 1940	64
1896	Rice, Walter Henry	Boston	April 19, 1940	68
1911	Riggs, Austen Fox	Stockbridge	March 5, 1940	64
1929	Roberts, Sumner Mead	Hyannis	November 19, 1939	41
1912	Salles, John Murray	New Bedford	November 26, 1939	55
1905	Scanlan, Thomas John	West Roxbury	October 18, 1939	67
1906	†Schmidt, Richard Deidrich	Dorchester	December 18, 1939	68
1884	†Sparhawk, Clement Willis	Salem	October 22, 1939	85
1891	Stiles, Fred Merritt	Waltham	May 10, 1940	76
1932	Stivers, George Lincoln	Tucson, Ariz.	November 26, 1939	62
1914	Thompson, Charles Arthur	Newton	February 3, 1940	67
1870	†Thompson, Frederick Henry	Fitchburg	December 14, 1939	95
1916	Van Gaasbeek, George Henry	Springfield	January 22, 1940	76
1882	†Vickery, Herman Frank	Brookline	February 22, 1940	84
1914	†Wagner, Emma Juliet	West Somerville	June 12, 1939	76
1904	Walcott, Henry Joel	Concord	April 17, 1940	67
1933	Weinger, Morris Aaron	Lynn	January 27, 1940	40
1922	White, Frank Dunster	Milford	August 28, 1939	63
1935	Wilkinson, William D.	Boston	September 23, 1939	41
1910	Wright, William Francis	Miami, Fla.	April 11, 1940	59
1914	Young, Edward Wallace	New Bedford	April 16, 1940	53

†Retired fellow.

Total number of deaths of active fellows	81
Total number of deaths of retired fellows	18
Grand total	99

Officers for 1940-1941

Elected by the Council, May 21, 1940

President: Walter G. Phippen, Salem, 31 Chestnut Street.

Vice-President: Frank R. Ober, Boston, 234 Marlboro Street.

Secretary: Alexander S. Begg, West Roxbury. Office, Boston, 8 Fenway.

Treasurer: Charles S. Butler, Boston, 257 Newbury Street.

Orator: A. Warren Stearns, Billerica. Office, Boston, 520 Commonwealth Avenue.

Standing Committees for 1940-1941

Elected by the Council, May 21, 1940

Publications — Established 1825.

	Date of appointment
F. H. Lahey	June 6, 1933 (appointed chairman February 7, 1940)
R. M. Smith	June 6, 1933
J. P. O'Hare	June 9, 1936
Conrad Wesselhoeft	June 2, 1937
W. B. Breed	February 7, 1940

Arrangements — Established 1849.

E. J. O'Brien	June 9, 1936 (appointed chairman May 21, 1940)
W. T. O'Halloran	June 2, 1937
J. A. Halsted	June 1, 1938
G. P. Sturgis	June 7, 1939
H. H. Faxon	May 21, 1940

Ethics and Discipline — Established 1871.

R. L. DeNormandie	June 11, 1929 (appointed chairman June 1, 1938)
R. R. Stratton	June 9, 1936
W. J. Brickley	February 3, 1937
A. G. Rice	June 1, 1938
F. R. Jouett	May 21, 1940

Medical Education and Medical Diplomas — Established 1881.

J. P. Monks	June 7, 1939 (appointed chairman May 21, 1940)
A. W. Stearns	June 9, 1936
A. R. Gardner	June 1, 1938
G. D. Henderson	June 1, 1938
L. S. McKittrick	May 21, 1940

State and National Legislation — Established 1894.

E. L. Merritt	May 21, 1940 (chairman)
D. L. Lionberger	June 4, 1935
B. F. Conley	June 2, 1937
C. A. Robinson	June 1, 1938
E. M. Chapman	June 7, 1939

Membership — Established 1897.

G. C. Caner	June 17, 1930 (appointed chairman May 21, 1940)
J. E. Fish	June 17, 1930
H. F. Newton	June 9, 1931
P. H. Leavitt	June 1, 1938
A. W. Reggio	May 21, 1940

Public Health — Established 1912.

F. P. Denny	June 1, 1938 (appointed chairman June 7, 1939)
Gerald Hoeffel	June 17, 1930

S. C. Dalrymple	June 4, 1935
H. L. Lombard	June 4, 1935
H. F. Day	June 7, 1939

MEDICAL DEFENSE — Established 1927.

A. W. Allen	June 7, 1927 (appointed chairman June 7, 1939)
E. D. Gardner	June 7, 1927
F. B. Sweet	June 7, 1927
W. R. Morrison	June 9, 1936
Horatio Rogers	June 7, 1939

PERMANENT HOME — Established 1932.

W. H. Robey	February 24, 1937 (chairman)
C. G. Mixter	June 8, 1932
J. M. Birnie	June 8, 1932
C. S. Butler	June 4, 1935
E. C. Miller	June 4, 1935

FINANCIAL PLANNING AND BUDGET — Established 1938.

John Homans	June 2, 1938 (chairman)
E. L. Hunt	June 2, 1938
C. F. Wilinsky	June 2, 1938
E. J. O'Brien	June 2, 1938
P. P. Johnson	October 4, 1939

SPECIAL COMMITTEES FOR 1940-1941

CANCER — Established 1917.

Shields Warren, chairman; F. G. Balch, E. M. Daland, P. E. Truesdale, C. C. Simmons.

REPRESENTATIVES TO THE MASSACHUSETTS CENTRAL HEALTH COUNCIL

Barnstable: W. D. Kinney
Berkshire: R. J. Carpenter
Hampden: G. D. Henderson
Middlesex North: M. A. Tighe
Suffolk: R. B. Osgood
Worcester: E. C. Miller

PUBLIC EDUCATION (a subcommittee of the Committee on Public Health) — Established 1930.

F. P. Denny, chairman; Gerald Hoeffel, secretary; G. R. Minot, W. H. Robey, R. M. Smith, E. H. Place, C. C. Simmons, J. H. Pratt, H. W. Stevens, J. B. Ayer, H. P. Mosher, F. R. Ober, E. P. Joslin, J. D. Barney, H. L. Lombard.

PUBLIC RELATIONS — Established 1931. (One member appointed yearly by each district medical society; the president of the Society is chairman.)

BARNSTABLE DISTRICT MEDICAL SOCIETY
M. E. Champion, North Harwich.

BERKSHIRE DISTRICT MEDICAL SOCIETY
P. J. Sullivan, Dalton, 471 Main Street.

BRISTOL NORTH DISTRICT MEDICAL SOCIETY
F. H. Dunbar, Mansfield. Office, Boston, 43 Bay State Road.

BRISTOL SOUTH DISTRICT MEDICAL SOCIETY
A. J. Pothier, New Bedford, 720 County Street.

ESSEX NORTH DISTRICT MEDICAL SOCIETY
E. S. Bagnall, Groveland, 281 Main Street.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY
G. K. Fenn, Beverly, 19 Abbott Street.

FRANKLIN DISTRICT MEDICAL SOCIETY
H. G. Stetson, Greenfield, 39 Federal Street.

HAMPDEN DISTRICT MEDICAL SOCIETY
P. E. Gear, Holyoke, 188 Chestnut Street.

HAMPSHIRE DISTRICT MEDICAL SOCIETY
E. E. Thomas, Northampton, 59 West Street.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY
J. H. Blaisdell, Winchester. Office, Boston, 45 Bay State Road.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY
M. A. Tighe, Lowell, 9 Central Street.

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY
D. C. Dow, Cambridge, 1587 Massachusetts Avenue.

NORFOLK DISTRICT MEDICAL SOCIETY
F. P. McCarthy, Milton. Office, Boston, 371 Commonwealth Avenue.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY
W. G. Curtis, Wollaston, 10 Grand View Avenue.

PLYMOUTH DISTRICT MEDICAL SOCIETY
C. D. McCann, Brockton, 12 Cottage Street.

SUFFOLK DISTRICT MEDICAL SOCIETY
A. A. Hornor, Boston, 319 Longwood Avenue.

WORCESTER DISTRICT MEDICAL SOCIETY
C. A. Sparrow, Worcester, 73 Sagamore Road.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY
J. J. Curley, Leominster, 82 Main Street.

POSTGRADUATE INSTRUCTION — Established 1932.

F. R. Ober, chairman; L. E. Parkins, secretary; F. D. Adams, Roy Morgan, J. M. Birnie, H. L. Higgins, J. W. O'Connor, C. W. Blackett, Jr., R. B. Osgood, Reginald Fitz, A. S. Begg, C. S. Burwell, A. W. Stearns, W. H. Robey, R. N. Nye, H. D. Chadwick, C. M. Campbell, Lincoln Davis.

PHYSICAL THERAPY — Established 1935.

F. P. Lowry, chairman; R. B. Osgood, G. R. Minot.

COMMITTEE TO CONSIDER EXPERT TESTIMONY — Established 1936.

G. L. Schadt, chairman; David Cheever, J. J. Goodwin, F. P. McCarthy, H. C. Marble.

AUTOMOBILE INSURANCE CLAIMS — Established 1937.

H. C. Marble, chairman; H. M. Landesman, secretary; P. P. Henson.

CONVALESCENT CARE — Established 1938.

T. D. Jones, chairman; H. E. Gallup.

INDUSTRIAL HEALTH — Established 1939.

W. I. Clark, chairman; N. G. Monroe, L. R. Daniels, H. G. Murray, T. L. Shipman.

ARMY MEDICAL LIBRARY AND MUSEUM — Established 1939.

H. R. Viets, chairman; R. B. Osgood, Benjamin Spector.

COMMITTEE TO STUDY PRACTICE OF MEDICINE BY UNREGISTERED PERSONS — Established 1939.

Richard Dutton, chairman; B. F. Conley, E. F. Timmins.

TWENTY-FIVE VOTING MEMBERS IN THE ASSOCIATED HOSPITAL SERVICE CORPORATION — Established 1939.

B. H. Alton, E. S. Bagnall, G. M. Balboni, W. B. Breed, L. D. Chapin, H. F. Day, J. F. Donaldson, A. W. Dudley, J. M. Fallon, J. E. Flynn, A. R. Gardner, H. W. Godfrey, D. C. Halbersleben, J. A. Halsted, J. H. Lambert, A. A. Levi, A. E. Parkhurst, Helen S. Pittman, A. G. Rice, A. T. Ronan, F. W. Snow, G. L. Steele, R. P. Stetson, J. E. Talbot, E. L. Young.

COMMITTEE TO STUDY PROPOSALS FOR BUDGETING MEDICAL CARE — Established 1940.

T. H. Lanman, chairman; J. M. Birnie, J. C. McCann, M. A. Tighe, Shields Warren.

DELEGATES AND ALTERNATES TO THE HOUSE OF DELEGATES, AMERICAN MEDICAL ASSOCIATION FOR 1940-1941

DELEGATES	ALTERNATES
June 1, 1939 to June 1, 1941	
E. F. Cody, New Bedford	E. L. Merritt, Fall River
J. M. Birnie, Springfield	R. J. Carpenter, North Adams
R. H. Miller, Boston	Cadis Phipps, Brookline
June 1, 1940 to June 1, 1942	
D. D. Scannell, Jamaica Plain	E. S. Bagnall, Groveland
Dwight O'Hara, Waltham	E. L. Hunt, Worcester
C. E. Mongan, Somerville	M. A. Tighe, Lowell
W. G. Phippen, Salem	J. I. B. Vail, Hyannis

COUNCILORS FOR 1940-1941

ELECTED BY THE DISTRICT MEDICAL SOCIETIES AT THEIR ANNUAL MEETINGS, APRIL 15 TO MAY 15, 1940

BARNSTABLE

O. S. Simpson, Falmouth, 59 Locust St., V. P.
M. E. Champion, North Harwich, A. M. N. C.
D. E. Higgins, Cotuit, Main St., Sec.
W. D. Kinney, Osterville, M. N. C.
E. S. Osborne, West Dennis.

BERKSHIRE

J. W. Bunce, North Adams, 85 Main St., V. P.
J. J. Boland, Pittsfield, 334 North St.
V. P. Cummings, North Adams, 57 Main St.
I. S. F. Dodd, Pittsfield, 34 Fenn St.
H. J. Downey, Pittsfield, 184 North St., M. N. C.
C. F. Fasce, Pittsfield, 311 North St., A. M. N. C.
C. F. Kernan, Pittsfield, 184 North St.
G. S. Reynolds, Pittsfield, 7 North St., Sec.
P. J. Sullivan, Dalton, 471 Main St.

BRISTOL NORTH

R. M. Chambers, Taunton, Taunton State Hospital, V. P.
W. H. Allen, Mansfield, 70 North Main St., M. N. C.
F. H. Dunbar, Mansfield, Office Boston, 43 Bay State Rd., A. M. N. C.
F. V. Murphy, Attleboro, 79 Bank St.
J. L. Murphy, Taunton, 23 Cedar St.
W. H. Swift, Taunton, 141 High St., Sec.

BRISTOL SOUTH

H. E. Perry, New Bedford, 159 Cottage St., V. P.
G. W. Blood, Fall River, 82 New Boston Rd.
R. B. Butler, Fall River, 278 North Main St., A. M. N. C.
E. F. Cody, New Bedford, 105 South Sixth St., M. N. C.
J. A. Fournier, Fall River, 11 Choate St.
E. D. Gardner, New Bedford, 150 Cottage St.
F. M. Howes, New Bedford, 135 Cottage St.
E. L. Merritt, Fall River, 130 Rock St., C.
D. R. Mills, Edgartown.
A. H. Sterns, New Bedford, 31 Seventh St., Sec.
I. N. Tilden, Mattapoisett, Barstow St.
C. C. Tripp, New Bedford, 416 County St.
P. E. Truesdale, Fall River, 151 Rock St.

ESSEX NORTH

R. C. Norris, Methuen, 247 Broadway, V. P.
E. S. Bagnall, Groveland, 281 Main St., A. M. N. C.
R. V. Baketel, Methuen, 7 Hampshire St.
C. S. Benson, Haverhill, 30 Summer St.
E. H. Ganley, Methuen, 251 Broadway.
H. R. Kurth, Lawrence, 477 Essex St., Sec.
P. J. Look, Andover, 115 Main St.
L. C. Peirce, Newburyport, 279 High St.
G. L. Richardson, Haverhill, 94 Emerson St.
F. W. Snow, Newburyport, 24 Essex St., M. N. C.
T. N. Stone, Haverhill, 3 Washington Sq.
C. F. Warren, Amesbury, 1 School St.
C. A. Weiss, Lawrence, 160 Jackson St.

ESSEX SOUTH

Loring Grimes, Swampscott, 84 Humphrey St., V. P.
H. A. Boyle, Middleton, Essex County Tuberculosis Sanatorium.
N. P. Breed, Lynn, 9 Washington Sq.
C. L. Curtis, Salem, 101 Federal St.
J. F. Donaldson, Salem, 32 Lynde St.
R. E. Foss, Peabody, 125 Main St.
S. E. Golden, Beverly, 38 Ocean St.
R. P. Hallett, Gloucester, 63 Middle St.
P. P. Johnson, Beverly, 1 Monument Sq.
J. F. Jordan, Peabody, 76 Lynn St., A. M. N. C.
B. B. Mansfield, Ipswich, 4 Green St.
A. E. Parkhurst, Beverly, Monument Sq.
W. G. Phippen, Salem, 31 Chestnut St., President.
Horace Poirier, Salem, 197 Lafayette St., M. N. C.
E. D. Reynolds, Danvers, 48 High St.
J. R. Shaughnessy, Salem, 24½ Winter St., Sec.
R. E. Stone, North Beverly, 15 Lakeview Ave.
J. W. Trask, East Lynn, 90 Ocean St.
C. F. Twomey, East Lynn, 80 Ocean St.

FRANKLIN

A. H. Wright, Northfield, 111 Main St., V. P.
F. J. Barnard, Greenfield, 479 Main St., M. N. C.
H. L. Craft, Ashfield, Sec.
A. H. Ellis, Greenfield, 58 Federal St.
W. J. Pelletier, Turners Falls, 171 Ave. A, A. M. N. C.
H. G. Stetson, Greenfield, 39 Federal St., Ex-Pres.

HAMPDEN

J. B. Bigelow, Holyoke, 109 Suffolk St., V. P.
F. H. Allen, Holyoke, 16 Fairfield Ave.
T. S. Bacon, Springfield, 69 Maple St.
E. P. Bagg, Holyoke, 207 Elm St.
W. C. Barnes, Springfield, 146 Chestnut St., Sec.
J. M. Birnie, Springfield, 146 Chestnut St., Ex-Pres.
W. A. R. Chapin, Springfield, 121 Chestnut St.
J. L. Chereskin, Springfield, 333 Bridge St.
A. J. Douglas, Westfield, 93 Elm St.
E. C. Dubois, Springfield, 174 Buckingham St.
G. L. Gabler, Holyoke, 98 Suffolk St.
M. F. Gaynor, Springfield, 44 Chestnut St.
P. E. Gear, Holyoke, 188 Chestnut St.
Frederic Hagler, Springfield, 20 Maple St.
G. D. Henderson, Holyoke, 312 Maple St.
E. A. Knowlton, Holyoke, 207 Elm St.
M. W. Pearson, Ware, 19 Pleasant St.
A. G. Rice, Springfield, 33 School St.
G. L. Schadt, Springfield, 44 Chestnut St., M. N. C.
G. L. Steele, Springfield, 20 Maple St., A. M. N. C.

HAMPSHIRE

J. M. Murphy, Florence, 91 North Main St., V. P.
A. J. Bonneville, Hatfield, 60 Main St.
J. D. Collins, Northampton, 187 Main St., Sec.
L. N. Durgin, Amherst, 66 Amity St., M. N. C.
L. B. Pond, Easthampton, 115 Main St., A. M. N. C.
E. E. Thomas, Northampton, 59 West St.

MIDDLESEX EAST

G. R. Murphy, Melrose, 244 Main St., V. P.
C. R. Baisley, Reading, 46 Woburn St.
J. H. Blaisdell, Winchester, Office Boston, 45 Bay State Rd., A. M. N. C.
Richard Dutton, Wakefield, 33 Avon St.
E. M. Halligan, Reading, 37 Salem St.
J. H. Kerrigan, Stoneham, 481 Main St.
K. L. Maclachlan, Melrose, 1 Bellevue Ave., Sec.
R. W. Sheehy, Winchester, 21 Washington St.
R. R. Stratton, Melrose, 538 Lynn Fells Parkway, M. N. C.

MIDDLESEX NORTH

W. M. Collins, Lowell, 174 Central St., V. P.
M. L. Alling, Lowell, 9 Central St.
R. L. Drapeau, Lowell, 310 Merrimack St., Sec.
D. J. Ellison, Lowell, 8 Merrimack St.
F. L. Gage, Lowell, 9 Central St., M. N. C.
A. R. Gardner, Lowell, 16 Shattuck St.
G. A. Leahey, Lowell, 128 Merrimack St.
E. O. Tabor, Lowell, 16 Shattuck St.
M. A. Tighe, Lowell, 9 Central St., A. M. N. C.

MIDDLESEX SOUTH

Dwight O'Hara, Waltham, Office Boston, 5 Bay State Rd., V. P.
C. F. Atwood, Arlington, 821 Massachusetts Ave.
E. W. Barron, Malden, Office Boston, 20 Ash St.
W. B. Bartlett, Concord, 28 Monument St.
Harris Bass, Everett, 351 Broadway.
E. H. Bigelow, Framingham Center, 31 Pleasant St., Ex-Pres.
G. F. H. Bowers, Newton Highlands, 156 Woodward St.
R. W. Buck, Waban, Office Boston, 5 Bay State Rd.
E. J. Butler, Cambridge, 25 Garden St.
Richard Collins, Jr., Waltham, 826 Main St.
B. F. Conley, Malden, 51 Main St.
P. A. Consales, Belmont, Office Boston, 481 Beacon St.
D. F. Cummings, Natick, 12 East Central St.
C. H. Dalton, Somerville, 440 Broadway.
H. F. Day, Cambridge, Office Boston, 412 Beacon St.
C. L. Derick, Newton Highlands, Office Boston, 412 Beacon St.
J. E. Dodd, Framingham, 259 Union Ave.
A. W. Dudley, Cambridge, 1740 Massachusetts Ave., M. N. C.
E. R. Fleming, Medford, 322 Boston Ave.
*H. Q. Gallupe, Waltham, 751 Main St.
F. W. Gay, Malden, 105 Salem St.
H. G. Giddings, Newton Centre, Office Boston, 270 Commonwealth Ave.
H. W. Godfrey, Auburndale, 14 Hancock St.
B. I. Goldberg, Newton Centre, Office Boston, 481 Beacon St.
W. G. Grandison, Charlestown, 65 High St.
A. D. Guthrie, Medford, 408 Salem St.
A. M. Jackson, Everett, 512 Broadway.
A. A. Levi, Newton, Office Boston, 481 Beacon St., Sec.
F. P. Lowry, Newton, 313 Washington St.
A. N. Makechnie, Cambridge, 14 Upland Rd.
R. A. McCarty, Waltham, 751 Main St.
J. A. McLean, West Somerville, 16 Curtis St.
Edward Mellus, Newton, 15 Clements Rd.
J. C. Merriam, Framingham, 198 Union Ave.
C. E. Mongan, Somerville, 24 Central St., Ex-Pres.
J. P. Nelligan, Cambridge, 2336 Massachusetts Ave.
E. J. O'Brien, Brighton, Office Boston, 270 Commonwealth Ave., C.
L. S. Pilcher, Newton Centre, 43 Parker St.
Max Ritvo, Newton, Office Boston, 485 Commonwealth Ave.
E. S. A. Robinson, Newton Centre, Office Jamaica Plain, 375 South St.
E. F. Ryan, Maynard, 74 Main St.
E. J. Sawyer, Newton, 488 Centre St.
M. J. Schlesinger, Newton, Office Boston, 330 Brookline Ave.
W. N. Secord, Watertown, 128 Mt. Auburn St.
J. W. Sever, Cambridge, Office Boston, 321 Dartmouth St.
E. F. Sewall, Somerville, 380 Broadway.
E. W. Small, Belmont, 68 Leonard St.
H. P. Stevens, Cambridge, 1 Craigie St.
R. A. Taylor, Waltham, 550 Moody St.
H. W. Thayer, Newtonville, 355 Walnut St.
J. H. Townsend, Belmont, Office Boston, 319 Longwood Ave.
Fresenius Van Nüys, Weston, 338 Boston Post Rd., A. M. N. C.
R. H. Wells, Lexington, 1430 Massachusetts Ave.
M. W. White, Somerville, 21 Walnut St.
W. S. Whittemore, Cambridge, 3 Concord Ave.
Alfred Worcester, Waltham, 314 Bacon St., Ex-Pres.
Hovhannes Zovickian, Watertown, 528 Mt. Auburn St.

NORFOLK

F. P. McCarthy, Milton, Office Boston, 371 Commonwealth Ave., V. P.
J. D. Adams, Brookline, Office Boston, 43 Bay State Rd.
F. J. Bailey, Dorchester, 40 Hancock St.
J. R. Barry, West Roxbury, 1857 Center St.
Carl Bearse, Boston, 483 Beacon St.

*Interim appointment.

A. S. Begg, West Roxbury, Office Boston, 8 Fenway, Secretary.
M. I. Berman, Dorchester, 1071A Blue Hill Ave.
G. F. Blood, Roslindale, 20 Belgrade Ave.
F. S. Cruickshank, Brookline, 1247 Beacon St., Sec.
William Dameshek, Brookline, Office Boston, 371 Commonwealth Ave.
F. P. Denny, Brookline, 111 High St., C.
G. L. Doherty, West Roxbury, Office Boston, 466 Commonwealth Ave.
D. G. Eldridge, Dorchester, 15 Monadnock St.
J. J. Elliott, Roslindale, 4258 Washington St.
H. M. Emmons, Needham, Office Boston, 354 Commonwealth Ave.
J. C. V. Fisher, West Roxbury, Office Boston, 510 Commonwealth Ave.
Susannah Friedman, Roxbury, Office Boston, 485 Commonwealth Ave.
Maurice Gerstein, Brookline, 1894 Beacon St.
David Glunts, Roxbury, 2 Franklin Garden.
W. A. Griffin, Sharon, Office Boston, 311 Beacon St.
B. T. Guild, Dorchester, Office Boston, 475 Commonwealth Ave.
J. B. Hall, Roxbury, 60 Windsor St.
R. J. Heffernan, Jamaica Plain, Office Brookline, 1101 Beacon St.
Morris Ingall, Roxbury, Office Boston, 485 Commonwealth Ave.
H. J. Inglis, Chestnut Hill, Office Boston, 43 Bay State Rd., A. M. N. C.
I. R. Jankelson, Jamaica Plain, Office Boston, 483 Beacon St.
H. L. Johnson, West Roxbury, Office Boston, 520 Commonwealth Ave.
C. J. Kickham, Brookline, Office Boston, 524 Commonwealth Ave.
C. J. E. Kickham, Jamaica Plain, Office Boston, 12 Bay State Rd.
E. L. Kickham, Brookline, Office Boston, 270 Commonwealth Ave.
D. L. Lionberger, Dedham, Office Roslindale, 3 Conway St.
D. S. Luce, Canton, 553 Washington St.
D. L. Lynch, Roslindale, Office Boston, 245 State St.
T. F. P. Lyons, Milton, Office Boston, 270 Commonwealth Ave.
Charles Malone, Jamaica Plain, 46 St. John St.
F. J. Moran, Dedham, 395 Washington St.
M. W. O'Connell, West Roxbury, Office Boston, Boston City Hospital.
Frederick Reis, Jamaica Plain, Office Boston, 416 Huntington Ave.
S. A. Robins, Roxbury, Office Boston, 636 Beacon St.
S. M. Saltz, Roxbury, 294 Seaver St.
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REPORT ON MEDICAL PROGRESS

DIABETES MELLITUS*

ELLIOTT P. JOSLIN, M.D.†

BOSTON

TRAUMA AND DIABETES

A RECENT article[1] on traumatic diabetes disturbs me. I fear from reading it that the impression may get abroad that trauma is a not infrequent precursor of diabetes. The authors report two cases which may be summarized as follows:

CASE 1. A 26-year-old obese woman, under treatment for several months with injections of Antuitrin-S, whose grandmother was diabetic, was struck by a streetcar on March 4 and thrown to the ground, remaining unconscious for 45 minutes. A life-insurance examination had been passed 4 months previously, and repeated examinations of urine the last week before the accident showed it to be sugar-free, as it was immediately after the accident and again on March 6. A spinal puncture yielded 15 cc. of frank blood under normal pressure. The patient was given 1500 units of tetanus antitoxin and 50 cc. of 50 per cent glucose intravenously. On March 8 excessive thirst and frequency of urination began and the fasting blood sugar was 278 mg. per 100 cc. This finding was overlooked and the patient was discharged on March 18. The diagnosis was cerebral concussion and multiple lacerations of the scalp.

Subsequently polydipsia, polyuria, a weight loss of 32 pounds, weakness and glycosuria developed. Having shown premonitory symptoms of acidosis for a week, the patient re-entered the hospital April 16 in deep coma, and died 14 hours later.

The medical examiner reported the presence of old, infiltrated blood in the deep layers of the galea. The brain was normal on external examination, and there was no evidence of surface injuries. The vessels at the base of the brain were normal and the ventricles normal in outline and clear, and there was no evidence of injury or disease in the fourth ventricle. The chest and abdominal cavities were normal, and microscopic examination of the organs, including the pancreas, revealed no abnormalities. No mention is made of microscopic examination of the brain.

CASE 2. A 14-year-old boy with negative diabetic heredity at the age of 5 years sustained a fractured skull and remained in a hospital for 5 weeks; at the age of 9 he had a second accident, with no definite findings, leaving the hospital in good condition after a few weeks. Tonsillectomy was performed at 10 years, and a physical examination, including the urine, at 12 was normal. During the summer of 1937 an otitis media developed, but cleared up after 2 months. In January, 1938, there was polydipsia, polyuria and a weight loss of 10 pounds, and at the end of 10 days the patient entered the hospital in a semicomatose state. The authors write:

There are no predisposing factors, such as obesity or family history. Whether or not trauma played a part in the obscure etiology we cannot say, but it may be assumed that it did. The case is one of many that we encounter, of sudden onset of diabetes with no demonstrable causative factor, and it emphasizes the importance of further study of such cases.

Lommel[2] reviews and discusses the relation of trauma to diabetes; he inclines to the view that there is no reason to exclude a traumatic etiology merely because one cannot explain such a connection, particularly in the presence of a proved disposition to diabetes.

These two articles have led me to summarize my own views on this question, and some of the reasons for them. But first of all I report two cases which have occurred in my own experience.

R. T. (No. 5786), a 13-year-old boy, went fishing through the ice on January 2, 1927, and fell in, but was rescued by his setter dog. That night polyuria began, and on January 14 there was 9 per cent of sugar in the urine. On January 26 the urine was sugar-free and the blood sugar was normal, both before and after meals, but the diabetes persisted.

However, I too fell through the ice as a child and my Newfoundland dog played a role in the rescue, yet I did not develop diabetes. Consider the number of boys who yearly have a similar escapade and never contract the disease! In contrast is the following:

The patient (No. 13332), a 14-year-old Jewish boy, with diabetic heredity, was in perfect health on December 24, 1934, so far as he or his family knew. That night he slept without rising from bed. On Christmas Day there was no special excitement or careless eating, yet that night he rose six times, and 17 days later, when I first saw him, the urine contained 8.0 per cent sugar.

There was no accident here to cause diabetes. But suppose he had suffered an injury on Christmas morning: consider the inferences which might have been drawn!

So far as I can remember, no definite case in which I considered trauma a cause of diabetes has occurred among the approximately 19,000 patients with diabetes mellitus and glycosuria who have consulted me. I know of no surgeon who has postponed an operation on a patient because of the possibility that trauma would bring on diabetes. I know of no case in which diabetes has been con-

*Prepared with the assistance of Howard F. Root, M.D., and Alexander Marble, M.D.

†Medical director, George F. Baker Clinic, New England Deaconess Hospital, Boston.

sidered caused by cerebral shock, embolism or thrombosis. I know of no case in which diabetes has been caused by accidents in the course of college athletics, particularly football, and to fortify my opinion I have recently consulted Dr. Arlie V. Bock, of the Department of Hygiene of Harvard University. He[3] writes:

> So far as I can determine, no case of diabetes following trauma has occurred among athletes at Harvard. We have had many types of injury but no known injury of the pancreas, and whether such trauma might result in diabetes I do not know. You know there has been close medical supervision of athletes at Harvard for at least twenty-five years.

I know of no case in which Dr. Harvey Cushing reported diabetes following the development of a tumor in the brain, save those cases in which diabetes occurred in connection with acromegaly and basophilism and 2 cases out of over 200 with chromophobe adenoma. "What is very significant," according to Dr. Louise Eisenhardt,[4] "is that in Dr. Cushing's own long experience in operating for tumors of the hypophysis or third ventricle he found that such operations did not result in even a transient glycosuria."

Dr. Gilbert Horrax[5] writes:

> I can certainly confirm what Dr. Eisenhardt has told you regarding pituitary cases, both from my experience with Dr. Cushing's patients and from that with my own. Furthermore, I have been unable to find in my own records any instances of glycosuria in cases with tumors of the fourth ventricle or fourth-ventricle region. However, this much must be said regarding our present cases, that it would be perhaps impossible to tell definitely about a traumatic glycosuria from operation in this region, since we now start all these patients with intravenous glucose at the beginning of the operation, and then if necessary they can have a transfusion without any interruption of the flow of fluid. However, before this operative technic I know of no case in our series.

During the World War injuries constantly occurred on the battlefield, but German, French and American writers agreed that they did not result in diabetes.

We have on record in this office the signed statement of a medical observer of pugilistic contests, as follows:

> In my twenty-five years of experience in examining boys and men for both amateur and professional boxing throughout the United States, I have never come across one person who has ever had any symptoms or any knowledge on his part submitted to me by him that he had diabetes or had been suffering from diabetes. In all the injuries due to trauma I have never known one injury resulting from trauma that would in any way cause diabetes in any form or any symptoms which would pertain to that disease. During my years of experience, 90 per cent of the people who have participated in boxing exhibitions either in the amateur or professional ranks have had repeated examinations, and none have ever shown any signs or symptoms of diabetes in any of its forms.

In the article by Geiger and Benson[1] I am mentioned as follows:

> Joslin dismisses the question briefly, admitting that trauma may aggravate diabetes, but denying a causal relationship. He would discard reports in the older literature because of insufficient proof of previous nonexistence of diabetes. He quotes von Noorden's very decisive statement based on the experiences of the World War, which, he says, once and for all bury the question.

In the sixth edition of my book,[6] eight pages were devoted to the question of trauma and diabetes, and forty-four pages were given over to the same topic, with a critical review of the literature and sixty-one references, in the section written by me in *Trauma and Disease*.[7] Since my report there has appeared a volume entitled *Studies of Trauma and Carbohydrate Metabolism, with Special Reference to the Existence of Traumatic Diabetes* by Thomsen.[8] The conclusions reached by Thomsen in this monograph are decidedly against trauma as a cause of the disease. An abstract of this volume appeared as an editorial[9] in the *Journal of the American Medical Association*.

The recent work of Young,[10] based on that of Houssay, Evans and others, in which the injection of anterior pituitary extracts leads to diabetes in animals, ultimately resulted in showing that this leads to degeneration of the islands of Langerhans, thus preserving the unity of the diabetes.

It is true that 1 case of possible traumatic diabetes has come to my attention in addition to the 2 cited in my monograph.[6] This case was published by Stern.[11] These 3 cases in which the evidence supports a traumatic origin of diabetes are not absolutely convincing. They are based on the destruction of a large portion of the pancreas, the only cause for diabetes recognized by most observers. As a rule, when injury to the pancreas, theoretically sufficient in extent to cause diabetes, has occurred, death has been immediate.

In Stern's case the patient was hit in the left side and back by an automobile on August 12, 1926, suffered with severe pain in the abdomen for 6 days, and when about to leave the hospital on the eighth day went into severe shock and collapsed with agonizing pain in the abdomen. Laparotomy revealed a hemorrhagic pancreatitis with diffuse peritonitis and fat necrosis. Recovery occurred after a 10 months' stay in the hospital, during which time two large intra-abdominal abscesses were opened. Later a subphrenic abscess developed, which was drained. On August 21 the blood sugar was 0.128 mg. per 100 cc.; on August 24, 0.140; and on August 27, 0.160 mg. About November, 1929, severe diabetes developed, with a blood sugar of 308 mg. per 100 cc. and 4 per cent sugar in the urine, both of which with dieting returned to normal. The father of the patient had died of diabetes at the age of 84 years; a daughter at the age of 30 had meliturıa, believed to be due to fructose, but the other three children

were apparently free of diabetes. Insulin has been taken since March, 1930.

Is it surprising that with some 660,000 diabetic patients now alive in this country,[12] and approximately 2,400,000 other individuals in our midst whom we can confidently expect to come down with it before they die, one may discover glycosuria and even diabetes occasionally following the million, more or less, injuries annually which these pre-diabetic people undergo?

The Experimental Production of Diabetes, Its Prevention and Cure

In contrast to recent skeptical views regarding the preventability of diabetes and its more severe stages, like a fresh breeze comes the news of recent experiments by Best[13] and Lukens[14] indicating clearly the possibility in animals of preventing and even of curing diabetes, a view first enunciated by Naunyn[15] and later confirmed in Allen's laboratory by Copp and Barclay.[16] The method of Young,[10] who produced permanent diabetes in dogs by the injection of large amounts of anterior-pituitary extract obtained from beeves, has been employed in various laboratories and the resulting experimental conditions carefully studied. Campbell, Keenan and Best[17] showed that in such animals the pancreas was almost devoid of insulin and the islets were the seat of hydropic degeneration and atrophy. In the spring of 1940 Best[13] announced that if, at the same time that the injection of anterior-pituitary extract was given to the dog, injections of large amounts of insulin were administered, the diabetes failed to appear and that no destructive changes occurred in the islands of Langerhans. This was the first demonstration of a means of preventing diabetes by experimental methods. Its clinical application lies in the desirability and indication for prompt and aggressive treatment of the disease at the earliest possible moment, as Naunyn[15] stated.

Marked differences in the susceptibility to the development of diabetes after injection of anterior-pituitary extract have been known. Thus, cats and rats are extremely resistant to its effect, but if a part of the pancreas is first removed the injection of anterior-pituitary extract will lead to diabetes. The rat reacts with hypertrophy of the island cells, the cat at first with hydropic degeneration. Lukens,[14] using cats, was able to show various stages in the production of diabetes and its cure. Thus in the cat with part of the pancreas removed, injections of anterior-pituitary extract were employed using the intraperitoneal route. Large amounts of the extract are required, owing in part to the fact that the inflammatory reaction within the peritoneum undoubtedly destroys a considerable amount of the active substance injected. In the cat the degenerative changes in the islands of Langerhans are gradual in their development. They consist of hydropic degeneration, followed after some months by atrophy and disappearance of many of the islands. By the end of six months the cats are permanently diabetic. On the other hand, during the early months of the diabetogenic injections a section of the pancreas may be removed by surgical means and the hydropic degeneration clearly demonstrated. After the cessation of the injections and the development of permanent diabetes of one to four months' duration, if doses of insulin are given in sufficient amounts a later histological examination of the pancreas will show disappearance of the hydropic degeneration, the return of the island cells to normal and, thus, the cure of the diabetes.

The possibility of regeneration of the islands of Langerhans when hydropic degeneration has occurred seems to rest on the removal of excessive metabolic strain. Apparently the effect of the injection of large amounts of anterior-pituitary substance is exerted in the tissues generally, consisting of increased breakdown of glycogen and an increased formation of glucose from protein. This seems to demand an increasing production of insulin by the islands of Langerhans until, by overstrain, degenerative changes are produced. Lukens points to cases described by Copp and Barclay,[16] working with Allen years ago, in which, after removal of a portion of the pancreas, hydropic degeneration in the islands was observed. In certain animals, by means of proper dietary treatment or by injection of insulin they were able to observe disappearance of the hydropic degeneration. Thus, either by diet or by the use of insulin, the islands of Langerhans may be spared and recovery result.

The possible clinical significance of these discoveries is immeasurable. Some of the peculiarities of acromegalic diabetes seem clearly explained. Thus, that only a small percentage of acromegalic patients develop diabetes has its counterpart in the fact that there is observed not only a species difference but a great individual variation in susceptibility of animals to the effect of the injection of anterior-pituitary extract. The apparent disappearance of diabetes during certain stages of acromegaly seems to be due to the subsidence of an acute active period of hyperpituitarism, which lasts only long enough to produce hydropic degeneration and then ceases, allowing for regeneration of the islands. This also would explain the fact that in the pancreas of some acromegalic patients at autopsy no change is observed in the islands. The

question of insulin resistance in acromegaly has been a matter of difference of opinion. Clearly all degrees of severity of diabetes may be expected in different types and stages of acromegaly. Cases requiring as much as 360 units of insulin a day, such as that of Ulrich,[18] may within a few days or weeks become so mild as to require no insulin. Yet cases of diabetes long established, as in the cases described by Coggeshall and Root,[19] show the usual types of diabetes. In such cases the hypoglycemic effects of insulin are identical with those seen in most diabetic patients.

Shall all cases of extreme insulin resistance be regarded as possible examples of the hypersecretion of some diabetogenic substance from the anterior pituitary gland? In one recent case a young clergyman (Case 17162) during the course of a few months developed such a resistance to insulin that he required 2000 units to recover from coma, and thereafter required a daily dose of 700 to 1500 units. However, at the end of eight months his requirement had fallen to 100 units daily. In this case the most careful studies revealed no evidence of acromegaly or hyperpituitarism. Yet a clear lesson is to be derived from these experiments, particularly with reference to patients who have a temporary exacerbation of diabetes during the course of infection. Thus, a young woman (Case 18978) thirty-eight years old required 124 units of insulin a day during severe pneumonia, yet two months later, during which period the insulin dose had gradually been reduced as the urine became sugar free, the blood-sugar values were normal and the diabetes had apparently disappeared. Of course this does not mean recovery, but the case will be watched. The effectiveness of insulin, then, during periods of infection or other temporarily increased strain on the islands of Langerhans may be, not merely to avoid acidosis and coma, but actually to protect the individual by resting the islands of Langerhans, and thus not only to prevent further damage to the islands, but even to allow for their return to normal.

Hypoglycemia

Hypoglycemia due to insulin continues to be a problem of major importance in the treatment of diabetes. In our experience 75 or 80 per cent of diabetic patients need insulin to enable them to utilize a satisfactory diet; of the 500,000 or 600,000 in the United States, it seems certain that at least 25 per cent, and possibly 50 per cent or more, actually take insulin. With all these an insulin reaction is an ever-present possibility. Patients must be informed of this fact at the beginning of treatment: they must be taught the causes, nature, symptoms and treatment of hypoglycemia. The facts must be presented without causing undue alarm and apprehension; fear of a reaction should never lead any patient to deny himself the benefit to be gained from insulin.

Fortunately, most individuals with diabetes are middle-aged or elderly, and most of these have mild diabetes. Consequently the dosage of insulin is small and the chances of developing hypoglycemia correspondingly less. In addition, patients vary in their sensitivity to insulin, and fortunately it is characteristic of the middle-aged or elderly person with mild diabetes to be relatively insensitive to insulin and relatively tolerant of overdosage. Furthermore, most insulin reactions which occur in the course of the treatment of diabetes are mild and cause the patient relatively little inconvenience and no injury.

Despite these considerations it is true that some diabetic patients, particularly juveniles, are extremely sensitive to insulin, develop reactions with great ease, and may have symptoms of a most unpleasant and even alarming nature. Moreover, repeated, severe reactions in this relatively small group increase apprehension among patients at large, and serve to handicap not only the hypoglycemic offender himself but also other diabetic patients in obtaining and keeping jobs. No one can censure an employer who objects to the scene created in a busy office when a young diabetic employee suddenly develops a severe reaction with pugnaciousness, struggling, crying-out, convulsions and possibly unconsciousness. Only one rule can be established: severe reactions must not occur, or at least must not occur frequently. It is the duty and the responsibility of the patient, his family and his doctor to design treatment in such a way that severe reactions are reduced to the lowest possible minimum.

Reactions due to protamine-zinc insulin may vary considerably from those brought on by regular or crystalline insulin. With the latter varieties, which are rapid-acting, the onset of symptoms takes place in a few minutes and usually these consist of nervousness, sweating, tremor, faintness, rapid heart action and double vision. On the other hand, protamine-zinc insulin acts so slowly that the onset of hypoglycemia may be insidious. Reactions after an overdose of protamine-zinc insulin are commonest twelve to twenty-four hours after it has been given. If it has

been taken before breakfast, the commonest time for the reaction is between midnight and morning. Symptoms often include occipital headache, nausea and even vomiting, leading in some cases, therefore, to confusion in diagnosis with diabetic coma. My experience with protamine-zinc insulin has been the same as that reported by Lowrie and Foster,[20] who state that with their patients not only do reactions occur less frequently with protamine-zinc insulin than with regular insulin, but they are in general less severe.

Although there is no direct and absolute correlation between hypoglycemic symptoms and the level of the blood sugar, it is most uncommon to have symptoms unless the blood sugar falls to 0.07 mg. per 100 cc. or below. Most alleged cases of reactions at higher levels in reality represent false diagnoses, due either to the apprehension of the patient or to the fact that conditions other than hypoglycemia may cause any or all the characteristic symptoms. One exception to this rule may be granted: when the blood sugar falls with extreme rapidity from a high to a considerably lower level, symptoms of a reaction may at times be felt without true hypoglycemia's being present. The level of blood sugar at which symptoms appear varies from patient to patient, and in the same individual from time to time. Thus, cases may be encountered in which no symptoms are present despite a blood sugar as low as 30 mg. per 100 cc. It must be remembered that the blood-sugar methods in common use measure certain non-glucose-reducing substances which total from 15 to 25 mg. per 100 cc.; consequently, a value below 30 mg. per 100 cc. indicates that there is little or no true sugar in the blood.[21]

The important point in the diagnosis of hypoglycemia in an unconscious patient is to avoid confusion with diabetic coma. In the latter condition, large doses of insulin are life-saving; in the hypoglycemic patient they may well be fatal. The differential diagnosis is well known, and need not be repeated here. One cannot overemphasize the importance of seeking laboratory confirmation at once in doubtful cases; without knowledge of the blood-sugar level the physician may find himself treating the patient blindly and for the wrong condition.

It has now become well established that although body tissues in general may turn to protein and fat in time of need, nervous tissue has glucose as its sole food.[22] Consequently, when the blood sugar falls to extremely low levels and the brain is deprived of sugar, cellular starvation results. Such a condition is incompatible with maintenance of the integrity of nerve cells. If profound hypoglycemia is long maintained, irreversible changes take place.[23] If this stage is reached, even though the blood sugar may subsequently be raised to normal, permanent damage to the nervous system may result. It is the experience of those giving shock treatments for schizophrenia that a small group of individuals remain in coma following cessation of a given treatment, even though the blood sugar has been raised to normal or supernormal levels.[24] In such patients both the immediate and the distant prognoses are grave.

The changes observed in the brain in fatal cases of hypoglycemia consist chiefly of tiny hemorrhages, multiple small thrombi, perivascular extravasations, interstitial edema and cortical necrobiosis affecting the ganglion cells.[25, 26]

In the treatment of diabetic patients the common causes of reactions are overdosage with insulin, failure to take the usual amount of food at the regular time or to assimilate it (vomiting, diarrhea), and unusual exertion. These suggest in themselves the important points in prevention. Insulin is a powerful preparation which must not be employed thoughtlessly. Although the dosage used must be determined largely by the method of trial and error, this should be done carefully and intelligently. Due thought should be given to the possible sensitiveness of the individual to insulin. With some patients, and particularly with children, with severe diabetes it may be found necessary to allow glycosuria and mild hyperglycemia at certain times during the day in order to avoid reactions at others. In the treatment of all diabetic patients, diet is fundamental, but in the patient receiving insulin it is more than ever important that the diet be uniform from day to day. With so many factors which are beyond one's control it is imperative that this influence be kept constant. Patients soon learn that if they take more than their accustomed amount of exercise a reaction may result. Consequently, whenever a change is made in the routine of living the possible effect on the insulin requirement should be considered in advance. It is a surprise each year to see with how much less insulin and how much more food diabetic children can be treated in summer camps where they are exercising daily out-of-doors than in the winter when activity is of necessity restricted. Before a period of any

unusual exercise patients should take a light lunch yielding from 10 to 20 gm. of carbohydrate. Patients driving an automobile throughout the day should eat something every two hours. Patients receiving protamine-zinc insulin should have a light lunch at bedtime. These lunches, designed for the prevention of reactions, should consist of food which requires digestion so that the effect on the blood sugar may be a sustained rather than a temporary one.[27, 28]

Mild attacks of hypoglycemia respond to relatively small amounts of food. Ten grams of carbohydrate as furnished by orange juice, ginger ale, sugar or candy usually suffices to overcome a reaction in five or ten minutes. More severe reactions, or reactions due to protamine-zinc insulin, may require larger amounts of carbohydrate. In patients who are unconscious the injection of 20 cc. of 50 per cent glucose intravenously in a sterile buffered solution usually gives prompt relief. It is customary to terminate a shock treatment for schizophrenia by administering glucose solution by gavage. Glucose may be given in relatively large amounts in 5 per cent concentration subcutaneously or intravenously. Well-nourished individuals may respond to the subcutaneous injection of 0.5 cc. of adrenalin or pituitary extract.

The symptoms of hypoglycemia due to insulin are essentially the same as those due to hypoglycemia arising from other causes. Among the latter are undernutrition, exhausting physical activity, hypofunctioning of the pituitary, thyroid and adrenal glands, extensive disease of the liver, glycogen-storage (von Gierke's) disease and hyperinsulinism. The last-named cause may reflect the presence of an islet-cell adenoma or carcinoma of the pancreas or, some have thought, generalized hypertrophy and overactivity of islet tissue without an actual tumor.[29] In all these conditions the treatment of the acute attacks is the supplying of food. To prevent recurrence specific treatment must be directed toward the underlying cause.

The paper of Campbell, Graham and Robinson[30] is of special interest because the features observed in their 5 cases are characteristic of those encountered in the treatment of patients with hyperinsulinism. In Case 1 an islet-cell adenoma was removed with apparent cure without incident, whereas in Case 2 it was not until a second operation that a tumor was found in the head of the pancreas; its removal effected a cure. In Case 3, following removal of a tumor, the blood sugar became normal, but the patient remained emotionally unstable and irritable and her memory and speech continued to be faulty, presumably because of serious damage inflicted on the nervous system by the prolonged hypoglycemia prior to the operation. In Case 4 death took place on the third day after operation, at which splenectomy and partial resection of the pancreas were necessary in order to remove a tumor in the tail of the organ. In Case 5 no tumor was found and a resection of 34 gm. of the pancreas was carried out. The patient had suffered no recurrence of hypoglycemia in the 10 months following the operation.

In reporting detailed studies in a patient with hyperinsulinism, Conn[31] mentions two practical points. The type of glucose-tolerance curve depends on the type of diet taken in the few days just preceding the test; a diabetic-like response may be obtained if the carbohydrate intake has been low. Certain patients may be kept from extremely low levels and convulsions prevented if epinephrin is given twice daily subcutaneously.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 26271

PRESENTATION OF CASE

First Admission. A fifty-seven-year-old, Canadian-born, retired broker entered the hospital complaining of swelling of the ankles and abdominal enlargement.

He suddenly became aware of these symptoms, and also of dyspnea on exertion, two months before entry, on the day the firm by which he was employed went into bankruptcy. There was no pain. He slept poorly because of nervousness but was comfortable with one pillow and had no nocturnal dyspnea. A slight cigarette cough had been present and unchanged for many years. His appetite had progressively failed, but though food was distasteful there had been no nausea or vomiting. During the two months he gained approximately 10 pounds. Various pills and salts prescribed by his physician had provided no appreciable relief. On two occasions the penis became swollen for a short time.

The family history was non-contributory. The past history recorded a hemorrhoidectomy seven years and an appendectomy four years previously. On very close questioning the following data regarding alcoholic consumption were elicited and were considered reliable: from 1912 to 1927, ten drinks a week (a drink was defined as 2 oz. of whiskey or gin); from 1927 to 1929, fourteen a week; from 1929 to 1932, six a week; and from 1932 to 1938, four a week. The consumption was usually concentrated on the week end.

Physical examination showed a well-developed and nourished, sun-tanned man, lying flat in bed without discomfort. Examination of the eyes was negative; the tongue was coated, the throat injected. The heart was not displaced and showed no evidence of enlargement. The apex impulse could be clearly seen and felt, and shifted with change in position. There was no Broadbent's sign. The sounds seemed muffled and faint; there were no murmurs. The blood pressure was 130 systolic, 100 diastolic. The rhythm was regular, and the rate 80. The aortic second sound was equal to the pulmonic second sound. The radial pulses were equal and synchronous, and the peripheral arteries were negative. The diaphragms were high and fixed. Dullness extended to just above the angle of the scapula on each side; the breath sounds were distant, and the spoken and whispered voice and the tactile fremitus reduced. Fine rales were heard over the dull areas. The abdomen was disproportionately large and showed shifting dullness, a fluid wave and early eversion of the umbilicus. The liver and spleen were not felt. There was bilateral pitting edema of the ankles and legs. Rectal examination showed a rosette of external hemorrhoids.

Six blood examinations showed hemoglobin concentrations ranging from 75 to 85 per cent (Tallqvist), red-cell counts from 4,600,000 to 5,000,000 and white-cell counts from 6000 to 13,000. The smear and differential counts were not remarkable. The urine showed specific gravities ranging from 1.004 to 1.030 and intermittent albuminuria (seven out of nineteen specimens). The sediment showed no red cells, variable numbers of white cells and, on occasion, hyaline casts. Three stools were guaiac negative. Two blood Hinton tests were negative. The nonprotein nitrogen was 20 mg. per 100 cc., the serum protein varied from 7.4 to 6.4 gm.; in the latter specimen the albumin was 3.7 gm. and the globulin 2.7 gm. The serum chlorides were equivalent to 93 cc. of N/10 sodium chloride. The Takata-Ara and formol-gel tests were negative.

An electrocardiogram showed a rate of 90, normal rhythm, a PR interval of 0.14 second, low T_1 and inverted T_3. The venous pressure in the left antecubital veins was equivalent to 60 mm. of water. The circulation time measured with saccharine (left arm to tongue) was 24.1 seconds. Abdominal paracenteses yielded abundant clear, straw-colored fluid, with a specific gravity of 1.018 and 1200 red blood cells and 250 white blood cells per cubic millimeter; a smear of the latter showed 9 per cent granulocytes, 38 per cent lymphocytes and 53 per cent large monocytes. Guinea-pig inoculation was negative for tuberculosis. Following the tap one examiner noted a poorly outlined mass in the epigastrium which descended with respiration; he could not feel the spleen.

X-ray examination showed a high diaphragm with good respiratory excursion. There was a small amount of fluid in each pleural cavity. The lower lung fields were poorly aerated. The heart was normal in size, the aorta slightly tortuous. Oblique views revealed nothing more. A gastrointestinal series, including an examination for esophageal varices, was negative. A barium enema showed scattered diverticulums throughout the colon. A flat abdominal plate showed an area of calcification, 0.5 cm. in diameter, in the lower pole of the left kidney. A confirmatory intravenous pyelo-

gram showed prompt excretion of the dye on both sides and localized the stone in the left lower calyx.

In the hospital, despite fairly good response to Salyrgan and Mercupurin, the ascites tended to reaccumulate. Peritoneoscopy showed no evidence of malignancy. The liver was normal in color, the surface smooth. Six weeks after entry an exploratory laparotomy was performed with some reluctance on the part of the surgeon. The liver was considerably enlarged but smooth. A biopsy was taken. Careful exploration of the stomach and colon revealed nothing, although adhesions from the previous appendectomy prevented palpation in the right lower quadrant. Pathological examination of the liver showed marked congestion and some atrophy of the liver cords in the centers of the lobule but no evidence of cirrhosis. One month later the patient was discharged with a clinical diagnosis of questionable cirrhosis.

Subsequent Admissions. During the next two years the patient re-entered the hospital twenty-five times. The majority of these visits were overnight entries to the Emergency Ward for tapping, but on four occasions he spent significant lengths of time on the wards. Despite Salyrgan every three days, tapping was necessary at least once a month and almost invariably temporarily relieved his symptoms.

Innumerable laboratory determinations were carried out. The hemoglobin concentrations and red-blood-cell counts fluctuated between 80 and 100 per cent and 4,000,000 and 5,000,000 respectively. The majority of white-cell counts fell between 6000 and 10,000 and the blood smears were never remarkable. The ascitic fluid never changed significantly in character, with specific gravities ranging from 1.011 to 1.018. Numerous examinations for tumor cells were negative. Van den Bergh tests hovered around 2 mg. per 100 cc. Bromsulfaleins ranged from 35 per cent retention on the second entry to 20 per cent on the fourth, basal metabolic rates from –4 to –21 per cent. Fifty serum-protein determinations varied from 4.79 to 7.4 gm. per 100 cc., with a slightly downward trend but a marked weekly variation. The serum albumin ranged from 2.9 to 3.7 gm. per 100 cc., and the globulin from 2.01 to 3.2 gm.; the albumin-globulin ratios varied from 0.98 to 1.72.

On several occasions, immediately after tapping, one or another member of the resident staff felt a non-tender midline epigastric mass which could be differentiated from the liver. Diagrams indicate that it was somewhat sausage-shaped and extended two thirds of the way from the ensiform to the umbilicus. The liver was frequently felt, the spleen never. Enlarged veins, which filled from below after milking, gradually became apparent on both sides of the abdomen. Nine months after the original x-ray examination a repeat gastrointestinal series demonstrated definite esophageal varices. Repeated chest plates showed fluctuations in the amount of pleural fluid, no change in the size of the heart and no evidence of a mediastinal tumor. Another electrocardiogram showed normal rhythm, a rate of 105, a PR interval of 0.14 second, low T_1, slightly inverted T_2 and T_3, low voltage in all leads and slight left-axis deviation.

Repeated transfusions with blood and with concentrated ascitic fluid failed to raise the serum-protein level significantly and to delay the reappearance of ascites. He gradually grew more and more emaciated, but the hemoglobin concentration and red-cell count remained within normal limits.

Final Admission (twenty-five months after the first symptom). The patient returned for abdominal paracentesis. Ten liters were removed with considerable relief, but he was still markedly dyspneic and examination revealed a large pleural effusion on the right side. Two thousand cubic centimeters of yellow, slightly cloudy fluid was removed. He failed to rally, became gradually disoriented, finally comatose and died two weeks after admission.

Differential Diagnosis

Dr. Chester S. Keefer*: The diagnosis in the present case must explain the recurrent ascites in a fifty-seven-year-old man who had an enlarged, smooth liver and a heart of normal size. When these signs co-exist there are two main conditions to consider: concretio cordis and thrombosis of the hepatic veins. Before discussing these two conditions, it is well to review the important features of the case.

In brief, the patient first noticed swelling of the abdomen and legs two months before entry to the hospital. At the time of admission, examination showed: ascites, a normal heart without signs of fixation, and edema of the ankles and legs. The blood, the plasma proteins and the venous pressure in the arms were normal. The urine showed intermittent albuminuria, and the ascitic fluid had the characteristics of a transudate. A mass which descended with respiration was felt in the epigastrium. X-ray films showed a small amount of fluid in both pleural cavities. A gastrointestinal series was negative except for diverticulosis, and there were no signs of esophageal varices. There was a stone in the left kidney.

Exploratory laparotomy revealed a large liver,

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which in a biopsied specimen showed evidence of chronic passive congestion. No masses or other abnormalities were discovered.

The course of the illness was of interest in that he had rapidly recurring ascites over a period of twenty-five months, and in that the signs of obstruction of the inferior vena cava, as well as of portal obstruction, became more distinctive, as evidenced by the dilated veins on both sides of the abdomen and the appearance of esophageal varices. Later, a massive pleural effusion developed and the patient died.

At the beginning of the discussion, I stated that recurrent ascites with an enlarged, smooth liver and a heart of normal size always suggest concretio cordis or thrombosis of the hepatic veins, especially when one knows, as in this case, that a section of the liver showed chronic passive congestion. There are good reasons for excluding concretio cordis, namely, the shift of the heart with change of position and the normal venous pressure in the arms. Therefore, I am going to dismiss this diagnosis.

The second condition, namely, thrombosis of the hepatic veins, is rare, but one must explain the enlarged liver with chronic passive congestion, the recurrent ascites, the evidence of portal hypertension (esophageal varices) and, finally, the edema of the legs. Let us review some of the facts concerning thrombosis of the hepatic veins. In spite of its relative infrequency, there are a number of features of this disorder which are exceedingly interesting. In the first place the diagnosis may be extremely difficult or, in some cases, impossible during life since the clinical picture frequently simulates that of portal obstruction. Indeed, it may be associated with the latter. A diagnosis may be entertained if a patient has recurrent ascites and an enlarged, smooth liver with a convex surface, without signs of concretio cordis. This is especially true if a biopsy of the liver is obtained and shows chronic passive congestion.

Portal obstruction will also produce recurrent ascites, but in such cases the liver is usually small unless there is a tumor which has invaded the intrahepatic branches of the portal vein, in which case the liver is enlarged and irregular. Obstruction of the portal vein alone will not explain the clinical picture in the present case.

There are several features of this case which cannot be explained on a basis of hepatic-vein occlusion alone, in particular the edema of the legs and the pleural effusion.

The edema of the legs and the dilated veins on the abdominal wall naturally suggest that the obstruction was in the upper third of the inferior vena cava as well as in the hepatic veins, that is, in that part of the cava where the hepatic veins enter.

The massive pleural effusion cannot be explained on the basis of obstruction of the inferior vena cava or hepatic veins. Naturally, this raises the question of the cause of pleural effusion in patients with liver disease. It may occur when there is an intercurrent tuberculosis of the pleura, a tumor of the liver with metastases to the pleuras or when there are low plasma proteins. In addition, it may be found when none of these conditions are present, especially when there is cirrhosis of the liver and hepatic insufficiency. This is a matter of great interest since Mann[1] found some years ago that partial excision of the liver was frequently followed by pleural effusion, an observation for which there was no adequate explanation. However, it is known that patients with hepatic insufficiency retain water; this has been ably demonstrated by Dr. Chester M. Jones.[2] It is possible, therefore, that the pleural effusion in this patient is associated, in some mysterious way, with the hepatic insufficiency.

If the clinical diagnosis of obstruction of the inferior vena cava and the hepatic veins is correct, then the question arises concerning its etiology. This condition has been observed in polycythemia vera, in tumors or infectious processes in the liver, in cirrhosis and in syphilis. There is no evidence for any of these conditions in this patient. Other cases have been described in which no definite cause for the thrombosis was found. In these it has been called endophlebitis obliterans hepatica. The process is frequently most conspicuous in the inferior cava about the mouths of the hepatic veins.

What about the possibility that a tumor had invaded the upper third of the inferior vena cava, thus causing this picture? This man was studied very carefully in an attempt to establish or exclude such a possibility. The tumors which invade the cava and occlude the hepatic veins as well are hypernephroma, tumors of the liver or of the testis and, rarely, tumors of the pancreas. At the time of operation no tumor was found. However, there is the recorded observation that a non-tender, midline, epigastric mass was felt which could be separated from the liver. Evidently this appeared after the exploratory operation and could be felt only after tapping. This may have been a tumor or a rolled-up, thickened omentum. In any event, I cannot explain it. It would seem unlikely, however, that a tumor of this size could have been overlooked at the time of operation, so that we do have to assume that it appeared following it. It

seems to me that the most likely diagnosis is an obstruction of the inferior vena cava and hepatic veins by a thrombus. I am, however, unable to make an etiologic diagnosis. My final diagnosis, then, reads: obstruction of the inferior vena cava and hepatic veins, with chronic passive congestion of the liver; esophageal varices; pleural effusion.

Dr. Tracy B. Mallory: Are there any questions or alternative diagnoses?

Dr. Howard C. Coggeshall: I was wondering about the spleen. Did the absence of splenomegaly influence you in making your diagnosis?

Dr. Keefer: The absence of splenomegaly is difficult to explain inasmuch as he had signs of portal obstruction, but in an individual fifty-seven years of age with portal obstruction the spleen is frequently not enlarged. Certainly you do not find it enlarged in more than about 50 per cent of the cases. I think there was enough evidence without splenomegaly to make the diagnosis of portal hypertension.

Clinical Diagnosis

Cirrhosis of liver.

Dr. Keefer's Diagnoses

Obstruction of the inferior vena cava and hepatic veins with chronic passive congestion of the liver.
Esophageal varices.
Pleural effusion.

Anatomical Diagnoses

Neurogenic fibrosarcoma of the inferior vena cava, occluding it from the mouths of the renal veins to the right auricle.
Cardiac cirrhosis of liver.
Esophageal varices.
Thrombosis of iliac veins and lower 3 cm. of vena cava.
Pleural effusion, right.
Ascites.
Operative scars: appendectomy, omentopexy, multiple paracentesis.

Pathological Discussion

Dr. Mallory: This case is a unique one, and I think Dr. Keefer has come as close as is reasonably possible. I can remember having a long discussion as to the clinical diagnosis at the time of the first biopsy of the liver, and my reasoning ran very close to that of Dr. Keefer. I argued that one had to place the obstruction in the cava close to or perhaps above the mouths of the hepatic veins and that if, as seemed to be the case, constrictive pericarditis could be ruled out, there must be thrombosis of the inferior vena cava of one sort or another. One other possibility also occurred to me; namely, that there might be a tumor of the left auricle so located that it would obstruct the mouth of the inferior without obstructing the mouth of the superior cava.

The autopsy presented a most confusing picture. The surgeon, who as has been said explored the

Figure 1.

The vena cava has been opened from behind showing the tumor extending upward to the point of section just below the auricle. The extracaval portion of the tumor lies to the left, partially covered by the aorta. The iliac vessels and the cava just above their union contain thrombi.

case reluctantly, did an omentopexy despite the fact that there was no cirrhosis, and as a result all the organs were rolled up in omentum and dissection was very difficult. It soon became apparent that the vena cava was distended to an enormous degree. It measured about 5 cm. in diameter and felt quite solid. On opening it we found a long sausage-shaped tumor, 20 cm. in length, which was lying for the most part free in the lumen but was attached to the lateral wall of the cava at about the level of the celiac axis (Fig. 1). At that point the tumor passed through the wall of the cava and out into the retroperitoneal tissues where another similar mass of almost but not quite equal size was present. So we had a dumbbell-shaped mass about half in and half outside of the cava. The tumor extended high enough to overlie the

mouths of the hepatic veins, which were stretched so taut that we had considerable difficulty in finding them, so I felt quite certain that there was obstruction at that point. The tumor actually extended 1.0 or 2.0 cm. into the right auricle. The liver, by the time of autopsy, had become somewhat smaller than it was when first observed. It was very slightly granular and was quite definitely tough. Microscopic examination of the liver post mortem showed well-marked cirrhosis of the so-called "cardiac type," which was not present in the biopsied specimen. The intervening two years of constant passive congestion explain the development of this type of cirrhosis during the period of observation. If I understood Dr. Keefer correctly he assumed that the obstruction to the hepatic veins occurred first and the caval obstruction later. The anatomical findings clearly indicate that the obstruction must have occurred in the reverse order. I believe that this is also borne out by the history, since the first really unequivocal evidence of hypertension in the portal circulation was the appearance of esophageal varices late in the disease, varices which were looked for and not found two years before. The tumor, itself, was a very slowly growing, spindle-cell sarcoma, I thought in all probability a neurogenic fibrosarcoma. Whether it was primary in the wall of the cava or started in one of the sympathetic ganglia in contact with the cava I cannot say.

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CASE 26272

PRESENTATION OF CASE

A twenty-year-old girl was admitted to the hospital complaining of dyspnea.

Two and a half years before admission the patient developed amenorrhea. She consulted her physician, who discovered the presence of diabetes and placed her on a diabetic diet. Her catamenia became normal again. She was sugar-free and apparently well under this regime for six months, when she became acutely ill, with severe headaches, dyspnea and anorexia. She became stuporous, and then comatose, and was placed in an outside hospital for a two-week period, where she was given a restricted diabetic diet with 20 units of insulin three times a day. On this regime she remained well until about two weeks before admission, when she developed a severe sore throat; Benedict's test for urine sugar became red, and the insulin dosage was raised to 25 units three times a day for a period of one week. Along with the sore throat there also occurred an elevated temperature, malaise and weakness. In addition her physician discovered high blood pressure. The sore throat persisted for one week, during which time an abscess in the right ear canal developed, which ruptured spontaneously. At about the same time she developed what she described as a papular rash over the face which persisted for some five days and then gradually disappeared. The next day, however, the ankles and both legs became edematous and she experienced chilly sensations. She had no other complaints until six days before admission when the peripheral edema increased and became evident over the body, face and upper extremities. Since that time the edema persisted. Five days before entry she developed a nonproductive cough, which prevented her from sleeping in the supine position. Symptomatic therapy by her physician failed to relieve the symptoms. She became thirsty and required large amounts of water. In spite of this, urinary excretion became less so that she urinated only four times in twenty-four hours and in small amounts on each occasion. The urine was sugar free, but for two days before hospital admission she noted that a white flocculation appeared while heating it. Three months before admission the patient weighed 123 pounds, at which level she had remained until one week before admission, when over the seven-day period she gained some 26 pounds. During her illness she did not complain of pain, night sweats or epistaxis.

As a child she had had chicken pox, pertussis, measles and tonsillitis, and about one cold with sore throat a year. There was a history of diabetes in a maternal aunt, a grandmother and a great-aunt. Her father, mother and two sisters were living and well, without symptoms of diabetes.

Physical examination revealed a pallid, puffy girl sitting bolt upright in bed, having marked dyspnea and orthopnea with a constant hacking, nonproductive cough. There was generalized pitting edema of the extremities, the trunk and the face. Examination of the retina revealed tortuosity of the arteries, out of proportion to her age, with marked arteriovenous nicking but no exudate or hemorrhage. The neck veins were moderately distended. The lungs were dull to percussion in both axillas and at both bases, with decreased tactile fremitus, breath sounds, whispered voice and spoken voice. There were multiple consonating rales heard about these levels on both sides; no friction sounds were audible. The heart was enlarged, the area of dullness extending 2 cm. beyond the midclavicular line in the fifth left interspace,

with a heaving, rapid apical impulse at a rate of 128. The sounds had a gallop rhythm. No definite murmurs were distinguished. The pulmonic second sound was slightly louder than the aortic second. The blood pressure was 168 systolic, 130 diastolic. The abdomen was edematous, with moderate ascites. The spleen was not felt, but the liver edge was palpable two fingerbreadths below the costal margin in the right midclavicular line, and was tender. The tourniquet test was positive.

The temperature was 100°F., the pulse 136, and the respirations 35.

Examination of the blood showed a red-cell count of 3,900,000 with 70 per cent hemoglobin, and a white-cell count of 9600 with 81 per cent polymorphonuclears, 18 per cent lymphocytes, and 1 per cent monocytes; the smear was normal. The urine was dark amber, with an acid reaction; the specific gravity was 1.029, with a ++++ test for albumin, a green sugar test and no diacetic acid or acetone; the sediment showed 10 to 20 red cells, 5 to 10 white cells and many finely and coarsely granular casts per high-power field. The carbon dioxide combining power was equivalent to 22.5 cc. of N/10 sodium bicarbonate per 100 cc. The total cholesterol was 185 mg. per 100 cc., the whole-blood nonprotein nitrogen 22 mg., and the blood sugar 130 mg.

The patient was given morphine for sedation and was rapidly digitalized both intravenously and by mouth, and fluids were permitted to aid in overcoming anuria. She was given insulin and was placed on a diabetic dietary regime. There was an apparent improvement in the general condition during the first twenty-four hours of her hospital stay, but she failed suddenly and expired on the second day. Immediately before death she was found to have had the following blood findings: carbon dioxide combining power, 22 cc. of N/10 sodium bicarbonate per 100 cc.; chlorides, 95.9 cc. of N/10 sodium chloride per liter; nonprotein nitrogen, 28 mg. per 100 cc.; protein 6.3 gm.; total base 156.7 milliequiv.

Differential Diagnosis

Dr. Bernard M. Jacobson: We are faced in this case with a combination of diabetes and hypertension, a situation that is not very commonly seen in a young person. It certainly is rare, moreover, for a person of this age to die, the way this girl died, of severe cardiovascular renal disease without more typical findings of glomerulonephritis. It is interesting that two and a half years before entry she developed amenorrhea and diabetes was found. One wonders momentarily whether this girl developed a Cushing's syndrome, but as subsequent events turned out, it is evident she did not. Her periods came back, and the diabetes was easily controlled. There is nothing on subsequent physical examination to suggest a Cushing's syndrome. Apparently over a period of two years the diabetes could not be called very severe. We do not know about the blood-sugar levels during this period. It was only two weeks before admission that the first mention was made of the presence of hypertension.

The present illness followed a respiratory infection and sore throat two weeks before admission, and six days before came the onset of edema, at first slight and then rapidly generalized, involving the trunk and extending up to the face. Orthopnea became marked twenty-four hours before entry. It is mentioned that although the urine was sugar-free, a white flocculation appeared on heating with Benedict's solution. That undoubtedly must mean the precipitation of urates due to concentrated urine, and certainly such a highly concentrated urine speaks for good kidney function, so far as excretion of solids goes. There was a remarkable gain of weight, — 26 pounds, — presumably due to edema, during the week before entry.

Physical examination revealed findings indicative of hypertension, hypertensive heart disease and subsequent myocardial insufficiency. The clinical signs of congestive heart failure were present, and the fact that the patient ran a very rapid pulse rate, with gallop rhythm, is certainly indicative of a very severe degree of myocardial insufficiency. When a person of this age goes into decompensation with abnormal rhythm it is indeed a grave omen. One additional finding besides those of congestive heart failure is the presence of edema of the upper part of the body. The patient was pallid and puffy, certainly findings that should attract our attention. They are not typical of congestive heart failure of the ordinary types, and they are important findings for further diagnosis. The note about the tourniquet test has no pathognomonic significance. We are not told about the presence of the free fluid in the chest by x-ray. I doubt from the physical signs if there could have been much, although there apparently was a slight pleural effusion. The blood showed mild secondary anemia and nothing remarkable in the white blood cells. The urine gave a ++++ test for albumin and other positive findings, which by themselves are not diagnostic. Certainly we see such urinary sediments and that much albumin in cases of severe cardiac decompensation without any significant degree of renal disease. It is interesting that the blood chemical findings were

normal. The serum protein was almost normal, and probably was slightly lowered merely by the albuminuria. We are not told whether the patient was given diuretics during the first twenty-four hours in the hospital. She probably was, with this much edema. Certainly the activity of the diabetes at the time of entrance to the hospital was not very great.

The rather sudden death suggests left-sided heart failure, possibly after the development of an abnormal rhythm. I do not believe that we learn anything more from the final blood determinations. The patient did not have diabetic acidosis or decompensated renal function. I am certain that the patient had diabetes mellitus, and hypertension, probably of long duration because of the size of the heart and because of the presence of definite arterial changes in the retinas, both tortuosity and marked arteriovenous nicking. There is no question she had myocardial insufficiency, which probably was the immediate cause of death.

We come to the question of what was going on in the kidneys. I think we cannot dismiss the type of edema that was present; its distribution in the upper part of the body, including the face, was very marked and noted by the patient and on physical examination on entry to the hospital. This type of edema suggests the question of whether this patient had some type of tubular change in the kidneys or whether the patient was in a nephrotic stage of glomerulonephritis or possibly was suffering from lipoid nephrosis. As I have said, the amounts of albumin in the urine and the sediment are still consistent with severe congestive heart failure. We have no history of acute glomerulonephritis in the past to suggest that this might be a subacute glomerulonephritis with edema. Certainly the amount of sediment in the urine is not enough to suggest acute nephritis, unless other urine examinations were done. Ten to 20 red cells and 5 to 10 white cells in the sediment are not consistent with an acute glomerulonephritis that came on two weeks after a respiratory infection and terminated in death.

DR. TRACY B. MALLORY: There are three urines recorded, with the specific gravity ranging from 1.028 to 1.033 and a ++++ test for albumin in all; there were 5 to 7 red cells on one occasion, and 10 to 20 on another, and they are reported as "crowded" on the third; white cells were always present.

DR. JACOBSON: Against acute glomerulonephritis is the fact that this patient must have had a hypertension of at least several years' duration prior to the onset of the terminal illness. Despite the absence of the history, is this possibly a subacute glomerulonephritis? I have no way of telling except for two factors. The patient had a normal blood cholesterol, and not a very markedly depressed serum protein. I think it is fair to say that this is not a typical nephrotic syndrome, despite the distribution of the edema. Once in a while we run into a nephrotic syndrome, somewhat imperfect, in the presence of other conditions in the kidney, such as amyloid disease. Certainly from the past history we have no evidence to suggest that this is amyloid disease.

I have mentioned the diagnoses that I think are certain. There is one other possibility which I see no way of proving, except to await Dr. Mallory's report. In the past few years there have been described in the literature several cases of a syndrome termed "diabetes mellitus, hypertension and intercapillary glomerulosclerosis." This was a syndrome first described by Kimmelstiel and Wilson[1] in 1936; one case has been reported by Derow, Altschule and Schlesinger[2] at the Beth Israel Hospital, Boston, and others by Newburger and Peters[3] at New Haven. All the cases reported had diabetes of several years' duration, all had hypertension, all had arterial changes in the retinas, and all had something pointing to disease in the kidneys. Some had the complete nephrotic syndrome, including low serum protein and high blood cholesterol values, but others lacked these characteristic blood chemical findings. At autopsy all these cases had the signs of long-standing hypertension and a rather high degree of sclerosis of the small arteries especially in the kidneys, with a very high degree of diffuse fibrosis of the central glomerular tufts with extension toward the periphery; many cases had, in addition, tubular changes of one sort or another. These were changes that these authors believed could be distinguished from those of ordinary active or healed subacute glomerulonephritis. I have never seen such a case, but it is my guess in this patient in order to explain the entire picture. I list my diagnoses as diabetes mellitus, hypertension, hypertensive heart disease with cardiac failure, and intercapillary glomerulosclerosis.

DR. EDWARD F. BLAND: Dr. Jacobson mentioned that he believed the hypertension must have been present for several years. I wonder why. Because of the eyegrounds?

DR. JACOBSON: Yes, and because the heart was considerably enlarged.

DR. BLAND: Of course the heart in young people can enlarge very quickly.

DR. MALLORY: A few years ago Dr. Earle M. Chapman studied patients with acute glomerulonephritis, and quite marked enlargement of the heart was demonstrable by x-ray in several which

disappeared in a few weeks as the renal disease subsided.

DR. BLAND: They see this picture at the Children's Hospital in patients with acute nephritis, but usually there is more evidence of acute nephritis.

DR. JACOBSON: We know that the hypertension existed for at least some time before she entered and before the edema appeared.

DR. BLAND: The patient did have a sore throat followed quickly by heart failure, so that we cannot forget diphtheria. It is very unlikely here, but we have to think of it.

DR. JAMES H. TOWNSEND: I have seen vascular disease in young diabetic patients, but it usually takes at least ten years of known diabetes to produce severe vascular disease at the age of twenty. I think it is probably fair to assume that the diabetes did not exist in this case more than two and a half years, because at this age diabetes makes itself manifest by symptoms soon after it begins. I have never seen extensive vascular disease develop in that length of time in a patient treated adequately, such as this patient seems to have been. I should be surprised if the postmortem showed generalized arteriosclerosis. My impressions from seeing her were that the diabetes probably had very little to do with the situation and that she developed an acute nephritis in the same way any non-diabetic patient might. Possibly because she was diabetic and since her resistance to infection was poor, she may have had a severer acute nephritis. Certainly all the edema was not cardiac, because she had it in the face and eyes and upper part of the body. The mode of death certainly was cardiac. She died with a dilated heart. Whether one can do that from acute nephritis I do not know, but I have the impression that one can die from cardiac failure in nephritis with edema, and this girl was terrifically edematous.

DR. PAUL D. WHITE: I have very little to add to Dr. Jacobson's excellent discussion. We were greatly disappointed in the patient's failure to recover from the congestive failure, for she was improving when she died suddenly. We could not explain the death, but thought of a possible abnormal heart rhythm.

It was evident from the history that the sore-throat episode was the vital factor precipitating the illness that ended in death three weeks later. We were inclined to think that the kidneys must have played an important role because of the clinical findings, despite the normality of the renal function tests.

One does see edema of the arms and face, especially unilateral, in congestive heart failure alone, but we thought that the kidneys entered the picture because of the high degree of bilateral edema of the face and arms.

CLINICAL DIAGNOSES

Diabetes mellitus.
Acute nephritis.
Cardiac dilatation, with decompensation.
Generalized anasarca.
Pulmonary edema.
Bilateral hydrothorax.
Ascites?

DR. JACOBSON'S DIAGNOSES

Diabetes mellitus.
Hypertension.
Hypertensive heart disease.
Acute cardiac failure.
Intercapillary glomerulosclerosis.

ANATOMICAL DIAGNOSES

(Diabetes mellitus).
Acute intracapillary glomerulonephritis.
Hypertrophy (slight) of the heart.
Acute cardiac failure.
Acute pulmonary edema.
Anasarca.

PATHOLOGICAL DISCUSSION

DR. MALLORY: This girl had two potentially lethal diseases, but I do not believe she died directly from either of them. The diabetes was perfectly controlled during the last few days of the illness, and she certainly did not die of renal insufficiency, at least not of uremia. But she did have acute nephritis. The kidneys were large and swollen, and nearly all the glomeruli showed definite intracapillary proliferative changes, probably not sufficiently severe, however, to produce uremia. Nevertheless, I think indirectly nephritis was the cause of death. Longcope[4] has very recently emphasized the frequent appearance of cardiac failure in patients with acute nephritis. Severe, almost paroxysmal, hypertensive symptoms may appear quite early in acute nephritis. He considers them at least in part responsible for the acute cardiac failure which he has frequently observed. Coming back to this patient there was a pleural effusion of 700 cc. on the right, and one of 200 cc. on the left. The lungs were extremely wet and heavy, and markedly edematous, and on anatomical grounds the best explanation of death is sudden left ventricular failure.

I am glad Dr. Jacobson brought up the question of intercapillary glomerulosclerosis, which should certainly be seriously considered in any

patient with the combination of hypertension, diabetes and a nephrotic syndrome. So far as I know, however, this condition has never been reported in a patient as young as this. The typical patients are usually around fifty.

DR. JACOBSON: I think the youngest one reported was forty.

DR. MALLORY: The age was the strongest point against that diagnosis, although it was a perfectly reasonable one to suggest.

DR. TOWNSEND: Did she show any generalized vascular disease?

DR. MALLORY: Virtually none. The heart was only slightly hypertrophied, weighing 350 gm. So I do not believe she had chronic hypertension.

DR. JACOBSON: I was thrown off by the eye-ground findings.

DR. TOWNSEND: Presumably they developed during the acute illness. I wonder if the nicking was not due, to a certain extent, to venous engorgement. She certainly had a great deal of generalized venous engorgement. The early history illustrates a mistake that is very often made with young diabetic patients in trying to manage them on a diet alone. They nearly always do the same thing in about six months: they will be in trouble of one type or another if one tries to manage them without insulin.

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RESULTS IN THE CAMPAIGN AGAINST SYPHILIS

The medical profession — and laymen as well — will be interested in the results of the campaign waged against syphilis under the sponsorship of Surgeon General Parran and the United States Public Health Service. The report* for the year 1939 reviews these results and indicates great progress.

The fact that 103,000 patients were discharged from clinics in 1939 as cured or with syphilis arrested, as compared with 78,000 in 1938, is most gratifying. Sixty per cent more persons were brought under treatment for the first time in 1939, and 65 per cent more treatments were given in clinics. Treatment and laboratory facilities increased between 30 and 85 per cent in the various states, and tests for syphilis showed a marked increase. More states set up separate divisions for venereal disease control, and more full-time health officers were employed. Nineteen states now require examination and serological tests for syphilis for marriage licenses, and fifteen require prenatal tests. Educational projects for physicians and laymen have been greatly expanded. Nine leading universities have established teaching centers for the training of physicians and nurses in the care of patients with venereal diseases, and research activities in various phases of the subject have been initiated or continued. Follow-up work has improved, and patients are made to feel the necessity for regular and adequate treatment. When figures are available, congenital infections will undoubtedly be found to be greatly decreased.

A combined attack against a major and incapacitating disease is being developed, and there is no reason for doubting that it will be successful, as have attacks in the past against smallpox, malaria, typhoid fever and yellow fever. With increased funds appropriated for the present year, and the experience and impetus given by the campaign thus far, it is reasonable to expect that a major victory will be achieved in this field of medicine.

*Progress in venereal disease control during the fiscal year 1939. *Ven. Dis. Inform.* 20:376-378, 1939.

WASHINGTONIAN HOSPITAL

On November 5, 1841, the present Washingtonian Hospital was organized as the Home for the Fallen, and on March 26, 1859, was incorporated as the Washingtonian Home and endowed as a place of treatment for alcoholic patients. It fulfilled a necessary function without any change of policy until 1939, when, due to the energy of Dr. Hilbert F. Day, an entirely new policy was projected which gradually became operative. The change was symbolized by the adoption of the name, Washingtonian Hospital. More concretely, a staff of visiting physicians in neurology and psychiatry made up of Drs. Leo Alexander, Wilfred Bloomberg, Robert E. Fleming and Merrill Moore was organized; daily visits are being made, and research and modern treatment have been instituted. The resident staff was altered, and now consists of Dr. Charles Parsons, superintendent, and Dr. Jos-

eph Thiman, resident physician. A woman nurse has been placed in charge of the nursing. The consulting staff is made up as follows: dermatology, Dr. Perry C. Baird, Jr.; internal medicine, Dr. Maurice B. Strauss; neurosurgery, Dr. William Jason Mixter; pathology, Dr. Harold A. McMahon; psychiatry, Dr. Abraham Myerson; surgery, Dr. Hilbert F. Day. Thus, medical activity of a modern type is being brought to bear on the problem of alcoholism.

The hospital asks for the support of the medical men of Metropolitan Boston in order that it may continue to grow and become an important factor in the understanding and treatment of alcoholism, which is now recognized as one of the major problems of modern society. Follow-up care will be given, and an outpatient department, which has the support of the Commissioner of Mental Health, Dr. Clifton T. Perkins, is in the process of development.

The aim of the hospital is to be of service to the alcoholic patient, to help the doctor in his care of these perplexing and difficult cases, and to advance the knowledge of the causes and treatment of alcoholism.

MEDICAL EPONYM

Charcot Joint

Jean Martin Charcot (1825–1893) described "quelques arthropathies qui paraissent dépendre d'une lésion du cerveau ou de la moelle épiniere [certain arthropathies apparently dependent on a lesion of the brain or spinal marrow]" in the *Archives de physiologie normale et pathologique* (1: 161–178, 379–400, 1868). A portion of the translation follows:

> We may summarize briefly the outstanding characteristics of the joint condition which we have observed in several ataxic patients. First, we again emphasize the absence of any external cause, traumatic or otherwise, any diathesis or constitutional influence to which it may be attributed. Syphilis, gonorrhea, the rheumatic or gouty diathesis, the prolonged effect of cold and moisture, either could not be blamed, or else were completely absent in the previous histories of our patients. So far as the localization is concerned, the arthropathy involved impartially either knee, sometimes the right, sometimes the left, and in one case the **left elbow. . . . The absence of fever,** redness and pain seemed an almost constant characteristic. . . . Swelling was always pronounced in the region of the joint itself, the principal cause being the accumulation of a certain amount of fluid in the synovial cavity; but at the same time the adjacent parts were constantly involved, sometimes over an extensive area. . . . Digital pressure left no pitting. In all cases the condition improved spontaneously and progressively . . . but nearly always there were . . . lesions which generally persisted, and which in one case were so marked as to cause a true dislocation of the joint.

R.W.B.

OBITUARY

GEORGE GRAY SEARS

1859–1940

George Gray Sears was born in Boston on October 22, 1859, and died there on May 28, 1940. His parents were George O. and Sarah G. (Richards) Sears. On February 2, 1904, he married Ruth Williams, of Boston. He had two daughters; one daughter, the wife of Dr. Richard Chute, of Boston, survives him. Except for his college days at Amherst, his eighty years of life were spent in Boston.

Additional facts in the chronology of his life are as follows: A.B. in 1880 and Sc.D. (hon.) in 1925, Amherst College; M.D. cum laude in 1885 and LL.D. in 1926, Harvard University; medical house pupil at the Massachusetts General Hospital, 1884–1885; district physician, Boston Dispensary, 1887–1889; outpatient physician, Carney Hospital, 1887–1889; visiting physician, Channing Home, 1891; physician to outpatients, 1893–1895, assistant visiting physician, 1895–1903, visiting physician, 1903–1919, trustee, 1918–1939, Boston City Hospital; trustee, Forsyth Dental Infirmary, 1924–; assistant, 1893–1897, instructor, 1897–1901, assistant professor, 1901–1911, associate professor, 1911, of clinical medicine, clinical professor of medicine, 1912–1918, professor of clinical medicine, 1918–1919, emeritus professor, 1919–, Harvard Medical School.

The facts of the life of George Gray Sears just enumerated form a silhouette but lack the details of line and color necessary to a portrait of the man as he lived and worked in the Boston community. What sort of a man was he? Not dynamic; not dramatic; never erratic; a quiet man of restrained mien, forceful, alertly watchful of his surroundings; never loquacious; able with few words, sometimes with a single word, to give the idea he wished conveyed; somewhat cynical at times, seemingly for its effect rather than expressive of his real self; tall, at no time either lean or

fat; with keen, kindly eyes and an evanescent smile playing about his mouth; a man that would be noticed in any gathering; a lovable, delightful companion to those who knew him well; filled with the wisdom that comes from observation and experience of men and matters, such was George Gray Sears. In his quiet, unobtrusive way he went through life busied with the practice of medicine, with teaching, with hospital visits, with trustee meetings, with interviews, all part of the work of a man whose concern broadly was with men, with institutions, with public affairs.

For forty-six years he labored at the Boston City Hospital in a period of phenomenal growth in number of patients, in size and quality of plant, in extension and improvement of the medical care of those who there sought cure of their disease and surcease from their suffering, and in the development of scientific study of disease. For twenty-six years he was a member of the clinical staff, for twenty-one years a trustee. When he retired as trustee, the senior staff presented him with an engraved testimonial signed by every member of the senior staff. This, among other things, it had to say of him:

> A gentle and lovable physician, a distinguished teacher, a wise administrator. . . . A gentleman learned and scholarly, a large part of whose busy life was diligently devoted to the hospital he loved. . . . He served faithfully through all the grades of the staff from the lowest to the highest. . . . His thorough knowledge of the professional problems of the hospital brought a fine balance to the Board of Trustees and was of invaluable aid in the solution of its difficulties.

At the Harvard Medical School he served actively for twenty-six years, progressively advancing from assistant to professor of clinical medicine. In sections he taught auscultation and percussion, on ward rounds he conducted clinical exercises, in the amphitheatre he held clinics or lectured. In showing to the students the way of making the correct diagnosis and directing the treatment of patients, he excelled.

In both institutions, as with patients, colleagues and friends, his part was to advise when asked for advice, to guide a movement already under way rather than to provoke its start. And yet he was not unprogressive, not lacking in initiative, not failing to look far into the future of medical developments. It was his way of getting things accomplished to give advice to the right person at the right time, to retard or to accelerate some movement by coming into its discussion at just the time his quiet, sage advice was needed, when, before this, he had listened and learned, put in an occasional query, but said very little. Few knew the many important things that were accomplished by him in this way, for he talked but little of what he had done; that they were many is made probable by a casual remark to patient or friend that occasionally, almost by accident, slipped out to indicate the part he had played in some development in Boston.

George Gray Sears was an ideal practitioner of medicine. He was an accurate observer, skillful in physical examination, inspiring in his patients that confidence which led them to divulge to him the intimate details of life and its reactions, facts so necessary for an understanding of disease as it affected that particular individual. Understanding his patients as he did and having their complete confidence, he became for them a wise adviser, not alone in matters of health but in all that concerned their daily lives. To his colleagues in medicine he rapidly became a valued consultant for their patients, and they sought his help and advice in the solution of many personal problems. To the younger physicians he was an example of the ideal practitioner.

A life filled with usefulness to patients, colleagues, friends and the community has ended. The memory of George Gray Sears long will remain as a benison to many.

HENRY A. CHRISTIAN.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

INFLUENZAL PNEUMONIA DURING PREGNANCY

Mrs. K., a twenty-one-year-old primipara, about thirty-six weeks pregnant, was seen in consultation on September 13, 1918, because of fever and prostration.

The family history was essentially negative. The patient gave a negative history. Catamenia began at twelve, were regular with a twenty-eight-day cycle, and lasted four days without pain. The last period had started on January 2, making the expected date of confinement October 9.

The pregnancy had been normal up to forty-eight hours before the patient was seen. At that

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

time she developed a cough and the temperature was 101°F., and the pulse 120.

Examination disclosed a well-developed and well-nourished young woman sitting up in bed. The respirations were embarrassed; the color was poor, the patient being slightly cyanotic. Rales were heard in both lungs. The heart was not enlarged; there were no murmurs. The blood pressure was 110 systolic. The uterus was enlarged to the size of an eight months' pregnancy, with the vertex presenting and the fetal heart audible. The uterus was not contracting. Although the patient did not seem seriously ill, a very guarded prognosis was given after a diagnosis of influenzal pneumonia had been made. It was advised that the patient be left alone, and be treated symptomatically and conservatively.

Three days later the membranes ruptured spontaneously and labor started immediately. The progress of labor was normal, and the patient was delivered normally, gas and oxygen being used as an analgesic and anesthetic. The baby was premature and rather slow to breathe, but lived. The patient's temperature came down to normal two days after delivery and remained so.

Comment. In the epidemic of 1918, influenza during pregnancy was a very serious complication. Some patients died undelivered, others were delivered and died. The question of inducing labor was frequently brought up only to be rejected, because it was believed that any operative procedure was contraindicated. The prognosis was grave and depended much more on the virulence of the infection than on any other single factor.

ARE VACATIONS NECESSARY?*

A well-known physician made the statement on one occasion that a man could do an excellent year's work in eleven months but not in twelve. This is simply a variation on the old theme that all work and no play makes Jack a total loss sooner or later, and what applies to Jack is equally true of Jill. The tired, nervous housewife and the hard driving business executive may both be headed for serious trouble if they do not occasionally call for "time out." Business and industry have to a great extent recognized the fact that not only is health served but efficiency is promoted by the granting of a vacation.

Before going any farther I should like to explain what a vacation really means. Anything which will give us mental and physical relaxation from the work which occupies us the year round can properly be called a vacation. It means forgetting our work, forgetting our cares and forgetting our scramble for greater wealth. Above all it should give us the opportunity to play and have some fun, both of which many adults have lost the ability to do, because of constant work, constant worry and constant tension over a period of many years. It should also mean a complete change of environment, because the cares and worries of everyday life drop away much more easily in new surroundings. And finally it should also mean a change in our usual type of activity. Much fun has been made of the mailman who spent his vacation hiking, the sailor who went boating, and the truck driver who took a cross-country automobile trip. On the other hand, the change in activity which is so advisable must not be carried to any unusual extremes. The young woman who has teetered through fifty weeks on four-inch French heels and suddenly puts on moccasins for long walks through woods and ravines, or the salesman who has spent most of his waking hours behind the wheel of an automobile and suddenly decides to climb seven mountains during his two-weeks-off may be in for a good deal of trouble because they have not taken into consideration that the human body may not be able to adapt itself to such violent changes of environment.

People who need vacations most frequently raise the objection either that they cannot afford one or that their work does not permit them to leave it for any length of time. Concerning the first objection, the matter of expense, aside from those unfortunate enough to barely eke out a living or slightly more, this question is usually not so forbidding as it appears. In the first place, with a little ingenuity, vacations involving very little expense can be arranged. And secondly, I am quite certain that the improvement in one's health which a proper vacation produces is not measurable in terms of dollars and cents. The amount of money thus saved on doctors' bills may help finance part of the vacation. To those who believe that their work cannot get along without them, I must repeat what has so often been said, that none of us are indispensable, particularly fifty-two weeks out of every year, and that the job we do will be a much better one for having been away from it for a while.

Many people, while agreeing in general that periodic vacations are highly desirable, nevertheless maintain that this is a luxury which they must forego, for a variety of reasons. This is an unfortunate point of view, and one that would be very easily changed could they but spend a few days as an unofficial observer in any doctor's office. There they would see patient after patient, in varying stages of ill health, unable to carry on their daily tasks efficiently. Many of these patients, to be sure, have organic diseases of different types and of various grades of severity. About 50 per cent of them, however, are organically quite sound. As their stories and symptoms unfold, the doctor realizes that their headaches and faulty digestions, their backaches and dizzy spells, are the direct result of life's stresses and strains and their worries and anxieties. With this group of patients, relief will be obtained not from the contents of a medicine bottle, which they all so earnestly seek. There is only one prescription that will give these individuals relief, and that prescription cannot be filled in any drugstore. That prescription is *rest.* Not only physical rest, but what is far more important, mental rest. I do not mean to imply that a periodic vacation will automatically restore this group of individuals to good health, but it will almost certainly result in marked and immediate improvement; it is at least one very important step on the road to recovery.

But why wait until we are rundown or ill before applying the remedy? Why not far more sensibly so arrange our affairs that a vacation is taken at least once a year, thereby minimizing our chances of becoming ill? That much illness can actually be prevented by vacations is very well known to physicians. This point was amusingly

*A "Green Lights to Health" broadcast given by Dr. Alfred Kranes, on June 11, 1940, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

illustrated several years ago in the advice given to a group of medical students by an eminent doctor. He advised them, when they were ready to begin practicing, not to start in the fall, because the summer vacations so improve people's health that the young doctor relying on new patients might become very discouraged. To avoid this discouragement he advised them to start in the spring when, because of the overwork and fatigue of the winter, patients would be more plentiful.

There is just one more point I should like to emphasize. Most of us, regardless of how interested we are in our work, develop a certain degree of irritability after being constantly on the job for any length of time. Petty things begin to annoy us; we snap at people for no apparent reason, and quarrels begin to arise at home, where heretofore all had been peaceful. We become difficult to live with and to get along with. The effect of a vacation on this state of affairs is usually miraculous. After a change of scenery and a rest, the world usually seems like a much better place in which to live, and our dispositions are much improved.

Q. Most of us really do not object to vacations, but the problem of expense is probably the most important single factor preventing the majority of people from taking one. Would you mind saying a little more about that?

A. "I can't afford it" is naturally what most people say whenever the doctor advises a vacation. But you must remember that we can all afford to keep well. Unfortunately people can only see what immediately concerns them, and in the vacation problem, it is the actual amount of money that a vacation involves. What they fail to see, because it is not imminent, is the cost of the illness that a vacation now and then may have prevented — not only the cost of doctors' bills and medicines, but the loss of wages that illness produces.

On the other hand, by budgeting income properly, as is done in Christmas Clubs, for example, sufficient money could be saved throughout the year to give many people a vacation they can afford. All it needs is a little planning.

Q. How long a vacation would you advise?

A. A little longer, I am afraid, than most people are able to take. The standard vacation seems to be about two weeks, and certainly two weeks out of fifty-two is the minimum that anyone ought to have. If it can possibly be done, four weeks once a year or two weeks twice a year is ideal. If two weeks is all that is available, it should be supplemented whenever possible by week ends away from our accustomed surroundings.

Q. Do you think it makes any difference what time of the year vacations should be taken?

A. Not the slightest. Whenever we can get the most fun and relaxation is the time to take our vacations. For many years, the summer has been the customary vacation season, but winter vacations are becoming increasingly popular. I think it will be very interesting to see whether the amount of disability due to respiratory infections during the cold months will be diminished as the winter-vacation fad increases.

Q. If, as you say, a vacation now and then is necessary for maintaining good health, does it make any difference where we go? To put it another way, is one place more healthful than another?

A. I do not believe so. The choice of where one goes depends on individual tastes. Whether it is the mountains or seashore, the city or the farm, actually makes very little difference provided we enjoy ourselves and get into new surroundings. A large city, for example, may not sound like a desirable vacation spot to most city dwellers, but to people who live on a farm the year round, it may be an ideal place to go. So far as concerns the old controversy about the mountains being more healthful than the seashore, or vice versa, there actually is nothing to it from a medical point of view. There may be occasional cases, in which, for special reasons, certain places are to be preferred, but for most of us my only advice is: go wherever you like and where you will have the greatest pleasure with the least responsibility.

Q. Are there any special precautions or advice that you think ought to be given to people going off on a vacation?

A. Well, it seems a bit queer to urge you in one breath to go away and enjoy yourself, and in the next breath to warn you about the trouble you may get into on a vacation. Nevertheless, in order that you may get the most out of it, perhaps it might be wise to mention several precautions.

The first thing that occurs to me is the common practice of sun bathing, and its dangers. I am not referring to the usual painful sunburn, which may, by the way, be quite serious. Although sunlight in repeated small doses may be quite beneficial, longer exposure, aside from the burn, may prove quite harmful. This is especially true in old people, in whom excessive exposure to the sun may result in serious strain on the heart. In any case it does them no good, despite all that is claimed for it and contrary to popular notions. I should advise all over fifty to protect themselves from the sun.

Another point which might be stressed again is the danger of overexertion. Here also the danger is not so much to young adults as to those above forty. To switch suddenly from a job behind a desk or counter to long-distance swimming or mountain climbing may be inviting serious trouble, and even at best, the aches, pains and fatigue following this unaccustomed effort do nothing to add to the joy of life, which was the primary purpose in going on a vacation.

DEATH

POPE — Ernest F. Pope, M.D., of Boston, died January 26. He was in his sixty-seventh year.

Dr. Pope received his degree from the University of Colorado School of Medicine in 1902, and had specialized in industrial surgery. He was a member of the Massachusetts Medical Society and the American Medical Association.

MISCELLANY

NOTES

It has been recently announced that the winner of the 1940 Mississippi Valley Medical Society Prize Essay is Dr. John W. Casey, of Boston, for his paper "The Study of the Use of Sulfapyridine and Sulfathiazole in Pneumonia, with Particular Reference to the Treatment of Pneumonia." Dr. Casey will present his essay at the sixth annual meeting of the society, to be held in Rock Island, Illinois, September 25, 26 and 27.

Recent dispatches list Dr. Laurence O'Shaughnessy, of London, as having been killed in action while serving with the Medical Corps of the British Army. Dr. O'Shaughnessy had gained world-wide fame for his operation of cardio-omentopexy, in which a portion of the omentum is brought up through the diaphragm, carried

through the pericardial sac and sutured to the heart in order to provide additional circulation in cases in which marked cardiac ischemia, due to coronary sclerosis and thrombosis, is present. He had quite recently visited the United States, and during his stay had presented a paper before the New England Heart Association.

CORRESPONDENCE

RESTORATION OF LICENSE

To the Editor: This is to inform you that in accordance with the vote of the Board of Registration in Medicine, the license of Dr. English N. McLaughlin, revoked on May 25, 1933, was restored on June 6, 1940.

STEPHEN RUSHMORE, M.D., *Secretary.*

State House,
Boston.

REPORT OF MEETING

WILLIAM HARVEY SOCIETY

At a regular meeting of the William Harvey Society held at the Beth Israel Hospital on April 5, Dr. Emil Novak spoke on "The Endocrine Influence of Certain Ovarian Neoplasms."

The speaker elucidated the subject on the basis of an embryological concept, recounting the normal phases of development of the germinal cells. It was hypothesized that groups of cells "nipped off" at various stages remain quiescent for some time and then become neoplastic and function according to their degree of differentiation at the time of segregation.

Tumors arising from cell rests of an early stage may occur in either the testis or ovary; they are markedly undifferentiated and have no functional activity. These are the dysgerminomas of the ovary or testis or the seminomas of the testis.

The first developmental stage is more or less toward a male phase with the female aspect being only evanescent. It is at this time that the testicular cords develop, and union with mesonephric structures occurs. Consequently, remnants of such cells later function to upset the intersexual balance in an individual toward maleness. Thus arrhenoblastomas may cause defeminization or masculinization.

As the germinal procession goes on there is a secondary wave of female development wherein the cells are grouped around the germ cells to form the future ovum and surrounding follicular epithelium. Overabundance of these cells may eventually be the cause of granulosal-cell tumors, which have the ability to produce estrogens and thus are feminizing tumors. The origin of both granulosal and thecal cells now appears to be mesenchymal in character rather than secondary to an ingrowth of germinal epithelium, so that thecomas and granulosal-cell tumors are properly part of one tumor group. This explains the usual picture of varying mixtures of the two types of cells in these tumors.

Dysgerminomas are prone to occur in those of poor sexual development, such as pseudohermaphrodites, despite their absence of sexual potentiality. Consequently, removal of these tumors has no effect on the underlying sexual balance.

Granulosal-cell cancers and thecomas both produce estrogen, and therefore have identical endocrine effects. Occasionally lutein-like cells may be formed, and if so, progestin effects, such as postmenopausal decidua, may be found. Naturally the endocrine effects of such tumors depend on the existing hormonal balance; thus the resultant manifestations are much more striking before puberty and after the menopause, when the amount of existing estrin is small.

In the very young, such neoplasms are a cause, although a relatively infrequent one, of precocious menstruation and development of secondary sex characteristics. The bleeding in such girls, however, is nonovulatory in contrast to that from extraovarian causes, such as small pituitary adenomas. During the reproductive phase of life, granulosal-cell cancer is one cause of menstrual disorders. There may be functional bleeding, normal menses or complete amenorrhea. The latter phenomenon is a result of continuous stimulation with estrin, a procedure which has been proved to be a means of producing amenorrhea. In the postmenopausal period there may be resumption of normal menstrual cycles with a certain amount of uterine rejuvenation; the breasts, however, do not share in this reversion. The typical manifestations of a second menopause on removal of granulosal-cell tumors in such women were offered as evidence that the pituitary stimulation which causes the symptoms of the menopause is secondary to the withdrawal of estrin.

Luteomas were considered to be luteinized granulosal-cell tumors or thecomas. They cause postmenopausal decidual changes of the uterus and have been known to exert a masculinizing effect in certain cases. An adrenal origin may explain this latter anomaly of function, since the resemblance of progestin, the adrenal cortical hormones and the androgens is well known.

The masculinizing tumors result from an attempt to form testicular structures in varying degree. Some are undifferentiated and simulate sarcomas, while others reproduce true cell types and have marked endocrine potentialities. Arrhenoblastomas may occur at any age, but usually appear during the third decade. The early manifestations are those of defeminization, such as amenorrhea, decrease of the breast tissue and loss of the female contour. Later, masculinization in the forms of a deep voice, enlarged larynx and clitoris, and hirsutism may ensue. Removal of the tumor causes definite but varying degrees of regression, with the menses and breast development showing the earliest changes. The masculine characteristics, especially the enlargement of the clitoris, retrace their course more slowly and often incompletely.

Adrenal rests may cause the same syndrome as that of the arrhenoblastomas, a fact which is not surprising when one considers the close embryological connection of the adrenal cortex and the ovarian medulla. In suspected cases, the pelvis should be examined, and the finding of an ovarian tumor, combined with the characteristic symptoms, should be taken as presumptive evidence of its etiologic relation.

NOTICES

ANNOUNCEMENT

CLITO R. DAMIANI, M.D., announces the removal of his office from 13 Kingsley Street, Allston, to 142 Craft Street, Newtonville.

UNITED STATES CIVIL SERVICE EXAMINATIONS

Chief Medical Officer $6500 a Year

Applications must be on file with the United States Civil Service Commission at Washington, District of Columbia, not later than July 8.

Applicants must have been graduated with an M.D. degree from a medical school of recognized standing; or must be a licentiate of the National Board of Medical Examiners. In addition they must have had certain highly responsible professional experience in the field of medicine, partly in a managerial or supervisory capacity. Applicants obtaining the highest ratings may also be requested to appear for an oral examination.

Full information regarding the examination and the application form may be obtained from the Secretary, Board of U. S. Civil Service Examiners, at any first- or second-class post office, or from the Civil Service Commission, Washington, District of Columbia.

SOCIETY MEETINGS AND CONFERENCES

SEPTEMBER 2–6 — American Congress of Physical Therapy. Page 862, issue of May 16.

OCTOBER 8–11 — American Public Health Association. Page 655, issue of April 11.

OCTOBER 11, 12 — Pan-American Congress of Ophthalmology. Page 898, issue of May 23.

OCTOBER 14–25 — 1940 Graduate Fortnight of the New York Academy of Medicine. Page 938, issue of May 30.

OCTOBER 21 — American Board of Internal Medicine. Page 369, issue of February 29.

JANUARY 4, 1941 — American Board of Obstetrics and Gynecology. Page 1064, issue of June 20.

APRIL 21–25, 1941 — American College of Physicians. Page 1065, issue of June 20.

DISTRICT MEDICAL SOCIETY

MIDDLESEX NORTH

JULY 31.

OCTOBER 30.

BOOK REVIEWS

A True History of the Terrible Epidemic Vulgarly Called the Throat Distemper Which Occurred in His Majesty's New England Colonies between the Years 1735 and 1740. By Ernest Caulfield, M.D. 8°, half cloth, 113 pp., with illustrations. New Haven, Conn.: published for the Beaumont Medical Club by the *Yale Journal of Biology & Medicine*, 1939. $2.50.

Dr. Caulfield will earn the gratitude of students and practitioners of medicine for this excellent, readable account of the terrible epidemic of "the throat distemper," which occurred in His Majesty's New England colonies between the years 1735 and 1740.

The author points out that, unfortunately for a scientific analysis, "the throat distemper" was a complicated epidemic, in that it consisted of a scarlet fever and two separate diphtheria epidemics. It is certain, the author continues to write, that diphtheria was endemic in the colonies for many years, particularly in Massachusetts and Connecticut, but not enough records are available to make a statement concerning its presence in New Hampshire. Scarlet fever was present certainly in Massachusetts and probably in New Hampshire, but there are no early records of it in Connecticut. The epidemic is carefully traced through towns in New Hampshire, Maine, Massachusetts and Connecticut, and reference is made to it in the states of New York and New Jersey.

One of the very noticeable characteristics of the epidemic was the occurrence of many deaths in the families of ministers and physicians. Many, in seeking for a cause, were "led to look beyond natural Causes to the Hand of God, to whom we are chiefly concerned to apply ourselves for the Removal of this awful Calamity." In Boston, on the other hand, "a consultation of Practitioners in Physick agree upon one important point: That the said Distemper proceeded from some occult Quality in the Air and not from any observable Infection communicated by Persons or Goods."

The student of medical history will especially appreciate the careful documentation of this book; it will be indispensable to the special student of infectious diseases.

Gardiner's Handbook of Skin Diseases. Revised by John Kinnear, T.D., M.D., M.R.C.P. (Edin.) Fourth edition. 12°, cloth, 239 pp., with 70 illustrations and 16 colored plates. Baltimore: Williams & Wilkins Company, 1939. $3.50.

This small handbook, which is well printed with large, readable type on good paper, does not present an adequate exposition of its subjects. It is somewhat more extensive than a quiz compend, but there are many fairly common, recent therapeutical procedures which are not mentioned. For instance, in psoriasis, the chrysarobin substitutes, such as anthralin, are not discussed.

The colored plates which were prepared from Dufay color photographs are quite good, but those which are obviously color photographs of moulages have a somewhat ancient appearance. The author's sketches indicating the histology of skin diseases are very naïve and charming in their simplicity, but therein lies some criticism because they barely more than indicate, somewhat vaguely, the outstanding microscopic characteristics. It is a pity that more effort to make them comprehensive was not expended.

It can hardly approach its aim as a handbook for the general practitioner unless the term handbook is used in a much more restricted sense than it usually is. It might do for students who are interested in no more than a quick sketch of some dermatoses.

Textbook of Medical Treatment by Various Authors. Edited by D. M. Dunlop, B.A. (Oxon.), M.D., F.R.C.P. (Edin.), L. S. P. Davidson, B.A. (Camb.), M.D., F.R.C.P. (Edin.), M.R.C.P. (Lond.), and J. W. McNee, D.S.O., D.Sc., M.D. (Glas.), F.R.C.P. (Lond.), with a foreword by A. J. Clark, B.A. (Camb.), M.D., D.P.H., F.R.C.P. (Lond.), F.R.S. 8°, cloth, 1127 pp., with 27 illustrations, and charts and tables. Baltimore: Williams & Wilkins Company, 1939. $8.00.

This is a textbook on medical treatment written by a group of Scotch physicians associated with the hospitals and schools in Edinburgh and Glasgow and bears the stamp of thoroughness and conciseness and the sense for the practical which characterize medicine in Scotland. It is a good book. It aims to consider the "management of the case" in the widest sense of the term and deals with therapy in its manifold applications. Furthermore, the authors "make an attempt to be extremely explicit in regard to the treatment recommended, in the hope that the directions given will suffice to enable a doctor without much previous experience to carry out the measures which have been described."

It is thoroughly up to date. Frequently, physiology and etiology are discussed briefly where these are essential to a thorough understanding of treatment. Diet, particularly, is stressed, and detailed lists and instruction are given with the individual disease entities. The authors recognize that a large portion of the physician's work is in relation to functional disease, and emphasize this aspect of therapeutics.

As one becomes more acquainted with this book it stands out as one of the better of the many textbooks on treatment.

Obstetrical Manikin Practice. By Lyle G. McNeile, M.D. 4°, cloth, 111 pp., with 38 illustrations. Baltimore: Williams & Wilkins Company, 1939. $2.00.

This small volume is designed for medical students who are taking a manikin course in obstetrics. Its purposes are to acquaint its readers with the anatomy of the female pelvis and to instruct them in the mechanism of normal birth and in the indications for and the technic of operative delivery. These aims are fulfilled in a satisfactory manner. The author's description of the mechanism of labor in vertex presentations, while departing in some respects from the classic accounts in standard textbooks, is probably correct, since it is based on reliable x-ray studies. The operative maneuvers advocated are those of sound modern obstetric practice.

The book may safely be placed in the hands of students, if their instructors will delete certain references to manual dilatation of the cervix — a dangerous procedure now justly in bad repute. In particular should the erasure be especially thorough where the suggestion is made that in performing a Braxton-Hicks version for placenta previa, it may be necessary to stretch the cervix sufficiently in this manner to secure a foot.

No mention is made of the increased height of the pelvis that is so often found in contractions of the male type and that is shown by lengthening of the pubotuberous diameter of Schuman. There are no illustrations of the soft parts making up the pelvic floor, which is of much more importance in the mechanism of normal labor than is the bony pelvis. In fact, since the whole book is devoted to the mechanics of normal and operative delivery and since a clear understanding of such mechanics depends on a series of accurate mental pictures in the minds of students, the illustrations might well have been increased in number.

A Text-Book of Occupational Diseases of the Skin. By Louis Schwartz, M.D. and Louis Tulipan, M.D. 8°, cloth, 799 pp., with 116 illustrations. Philadelphia: Lea & Febiger, 1939. $10.00.

Occupational dermatoses are assuming greater importance, and a book of this type should be a welcome addition to the library of many physicians. The subject is discussed from a general standpoint in the first 75 pages, in which are lists of substances for patch tests and a description of the methods of investigation of occupational skin diseases. Classification is discussed, and the matters of diagnosis, incidence and general methods of treatment also receive consideration.

The bulk of the book is devoted to the dermatoses caused by various agents, with many single chapters devoted to a particular substance. A large number of processes are discussed in detail, and information is provided about the risks attendant on the use of various substances. There are many valuable lists, such as cosmetic ingredients, dyes, fungicides, plants, resin plastics, woods and so forth, and there is much information about the various types of infections which possibly may be received in the course of work. Another section reviews the skin hazards in many different occupations. Finally, there is a chapter with data about many known irritants, and the most important industries in which they are employed. Thus the agent or the occupation may be reviewed by the physician.

There are numerous illustrations, and an excellent index. The book as a whole is well done and will be a valuable reference book for physicians doing part-time or full-time industrial work.

Recent Advances in Medical Science: A study of their social and economic implications. A Rede Lecture delivered before the University of Cambridge, on April 28, 1939. By Sir Edward Mellanby, K.C.B., M.D., F.R.C.P., F.R.S., K.H.P. 12°, paper, 62 pp. Cambridge, England: At the University Press, 1939. $.75.

In a very brief report, given as one of the Rede Lectures at the University of Cambridge, Sir Edward Mellanby, the energetic secretary of the Medical Research Council, has summed up some of the recent advances in medical science. The account is an extremely readable one but loses a good deal on account of its brevity.

Practical Obstetrics. By P. Brooke Bland, M.D. and Thaddeus L. Montgomery, M.D. Third revised edition. 4°, cloth, 877 pp., with 502 illustrations, including 27 colored plates. Philadelphia: F. A. Davis Company, 1939. $8.00.

This textbook, which is in reality a new edition of Bland's volume with Montgomery as co-author, is clearly and systematically written and adequately illustrated. Since it covers the subject in a rather cursory manner, it is more suited to students beginning their study of obstetrics than it is to practitioners seeking detailed information. It has two weaknesses. The section on obstetric pathology is considerably out of date, showing no changes from the edition of Bland's book which was published in 1932. It is doubtful if the description of the pathologic picture in pernicious vomiting or in eclampsia is sufficiently accurate to be acceptable today. The account of the medical and surgical complications of pregnancy does not appear adequate in view of the great amount of attention that these matters are now receiving.

Nutrition and Diet in Health and Disease. By James S. McLester, M.D. Third edition, entirely rewritten. 8°, cloth, 838 pp., with tables. Philadelphia and London: W. B. Saunders Company, 1939. $8.00.

There is no denying the fact that one of the fields of medical science which has attracted widespread attention is that of nutrition. Indeed, the advances which have been made in the past few years are so numerous that it is difficult for anyone to keep fully informed. With the appearance of the third edition of Dr. McLester's book, there is now available, in one volume, the information that every physician is anxious to obtain readily. This book has been entirely rewritten, and it is refreshing to find discussions of disordered physiology in preparing the reader for the outlines of the dietary treatment of disease. The material is clearly presented, and the text can be read with ease. This third edition should enjoy the same popularity as the first and second, and every well-informed physician will want to own a copy.

Historical Directory of State Health Departments in the United States of America. By Robert G. Paterson, Ph.D. 8°, paper, 68 pp. Columbus: The Ohio Public Health Association, 1939. $1.00.

The author has compiled a useful manual giving the name of the current executive in each state health department, with the names and titles of all executives since the establishment of the department. There is also a very valuable bibliography of all reports and publications of the various health departments. It is interesting to note that the Massachusetts Department of Public Health, established in 1869, was the first of its kind in the United States.